# The Journal
of
# Thoracic Surgery

Official Organ
*The American Association for Thoracic Surgery*

Editor
EVARTS A. GRAHAM†

Associate Editors

BRIAN BLADES
Washington, D. C

THOMAS H. BURFORD
St. Louis

ADVISORY EDITORIAL BOARD

ALFRED BLALOCK, Baltimore
EDWARD D. CHURCHILL, Boston
LEO ELOESSER, San Francisco
CAMERON HAIGHT, Ann Arbor
STUART W. HARRINGTON, Rochester, Minn
ROBERT M. JANES, Toronto
HERBERT C. MAIER, New York
RUDOLPH MATAS, New Orleans
RICHARD H. MEADE, Grand Rapids

ADVISORY EDITORIAL BOARD REPRESENTING THE SOCIETY OF THORACIC SURGEONS OF GREAT BRITAIN AND IRELAND

H. MORRISTON DAVIES, Ruthin, North Wales
R. C. BROCK, London
C. PRICE THOMAS, London

VOLUME 33

JANUARY—JUNE, 1957

ST. LOUIS
THE C. V. MOSBY COMPANY
1957

---

†Deceased March 4, 1957. Dr. Thomas H. Burford served as Acting Editor until May 6, when Dr. Emile Holman was elected by The American Association for Thoracic Surgery to succeed Dr. Graham.

VOLUME 33

COPYRIGHT, © 1957, BY
THE C. V. MOSBY COMPANY

*(All rights reserved)*

Printed in the
of America

The Journal of
Thoracic Surgery

Vol. 33 January, 1957 No. 1

## Original Communications

---

# A CLINICAL EVALUATION OF DECORTICATION

David H. Waterman, M.D., Sheldon E. Domm, M.D., and
William K. Rogers, M.D. (by invitation)
Knoxville, Tenn.

THE freeing of an imprisoned lung to obliterate persistent pleural space and, more important, to restore pulmonary function, has long been a surgical challenge. The first attempts by Fowler[1] and by Delorme[2, 3] in the 1890's were admittedly unsatisfactory as to surgical technique and results. Later, others,[4] particularly Eggers,[5] applied surgical principles to the operation that are still pertinent and valid. Without antibiotics and the present surgical safeguards, especially in anesthesia and blood replacement, the operation remained a most formidable one. Under Churchill's[6] direction in the Mediterranean Theater during the last war, Burford, Parker, and Samson[7] and others re-pioneered the field and introduced techniques and produced results that have revived interest in the procedure and made it accepted by all thoracic surgeons.

Quite naturally the first problem attacked by Fowler and by Delorme was empyema. Pleural infection continued to be the predominant challenge through the First World War and, in fact, until the Second. The dangers of a persistent pleural infection were well recognized. Obliteration of the space by various types of thoracoplasty, while accomplishing this end, left much to be desired. Decortication was applied in an attempt to obtain a more satisfactory surgical answer, primarily with the object in mind of obliterating pleural space.

The other pressing problem was hemothorax, particularly brought to the fore because of the large number of chest wounds of the past war. The concept of restoring a lung to function actually was initiated during this period when it was demonstrated that decortication would permit the injured soldier

---

From the Departments of Surgery of the Fort Sanders Presbyterian Hospital and the East Tennessee Tuberculosis Hospital, Knoxville, Tenn.

Read at the Thirty-sixth Annual Meeting of The American Association for Thoracic Surgery at Miami Beach, Fla., May 7 to 9, 1956.

to return to duty in a high percentage of instances. The technique was immediately applied widely in civilian practice as well, with comparable good results. Infection became less and less of a problem, though not infrequently complicating the hemothorax.

This was the era of pneumothorax therapy. Pneumothorax was theoretically and, in most cases practically, a reversible procedure. In too many, however, it was not, and space either persisted or "false re-expansion" resulted. In the first instance, decortication was seized upon as the solution to the problem, first by Mulvihill and Klopstock,[8] and then by many others. The results were most gratifying, with satisfactory obliteration of the space. Selective decortication,[9] leaving a plaque of peel over the area of disease, was used to render the danger of reactivation of foci less likely. Residual tuberculous disease in the lung at this time was rarely resected.

## MATERIAL

In the past eleven years, the authors have had an experience with 172 cases in which pulmonary decortication was performed either as a primary or a secondary procedure. Eighty-five per cent of these have been performed in the past six years. Of the 172 cases, 140 were operated upon by the authors, and 32 by other staff members of the East Tennessee Tuberculosis Hospital. It is the purpose of this report to analyze this group of cases as to the type of operative procedure performed, the indications for such procedures, the complications encountered, the mortality occurring, and the results obtained. Clinical evaluation in all cases, and pulmonary function studies, including bronchospirometry in a selected group, have been used to assay the results.

## OPERATIVE PROCEDURES

The types of operative procedures performed in the 172 cases are listed in Table I. Simple decortication alone was performed in 70 cases. Lobectomy was combined with decortication in 49, and segmental resection in 38. Included in this last figure are both dissection segmental resections and wedge procedures, giving a total of 87 patients in which pulmonary tissue was extirpated in conjunction with the decortication. Thoracoplasty was done in 10 patients having decortication, 8 of these being performed in connection with a relaxing thoracoplasty following resection. In such cases the pleural space was entered, the underlying lung decorticated, and the thorax tailored to fit the remaining space. The other 2 cases were in conjunction with Schede thoracoplasty done for long-standing empyema.

TABLE I. OPERATIVE PROCEDURES
172 CASES

| | |
|---|---|
| Decortication alone | 70 |
| with lobectomy | 49 |
| with segmental resection | 38 |
| with thoracoplasty | 10 |
| with diaphragmatic herniorrhaphy | 3 |
| with plication of diaphragm | 2 |

A diaphragmatic hernia was repaired in 3 cases, the decortication being a primary procedure and the hernia an incidental finding in one patient, and the reverse in the other 2.

In 2 cases of a high diaphragm, resulting from previous phrenic interruption remaining permanent rather than temporary, plication was performed to restore the diaphragm to a normal level and to permit the lung to expand in an adequate space. It was of interest that in both of these patients a markedly attenuated diaphragm, apparently showing no function whatsoever on fluoroscopy preoperatively, was restored to a limited excursion following the plication procedure.

## INDICATIONS

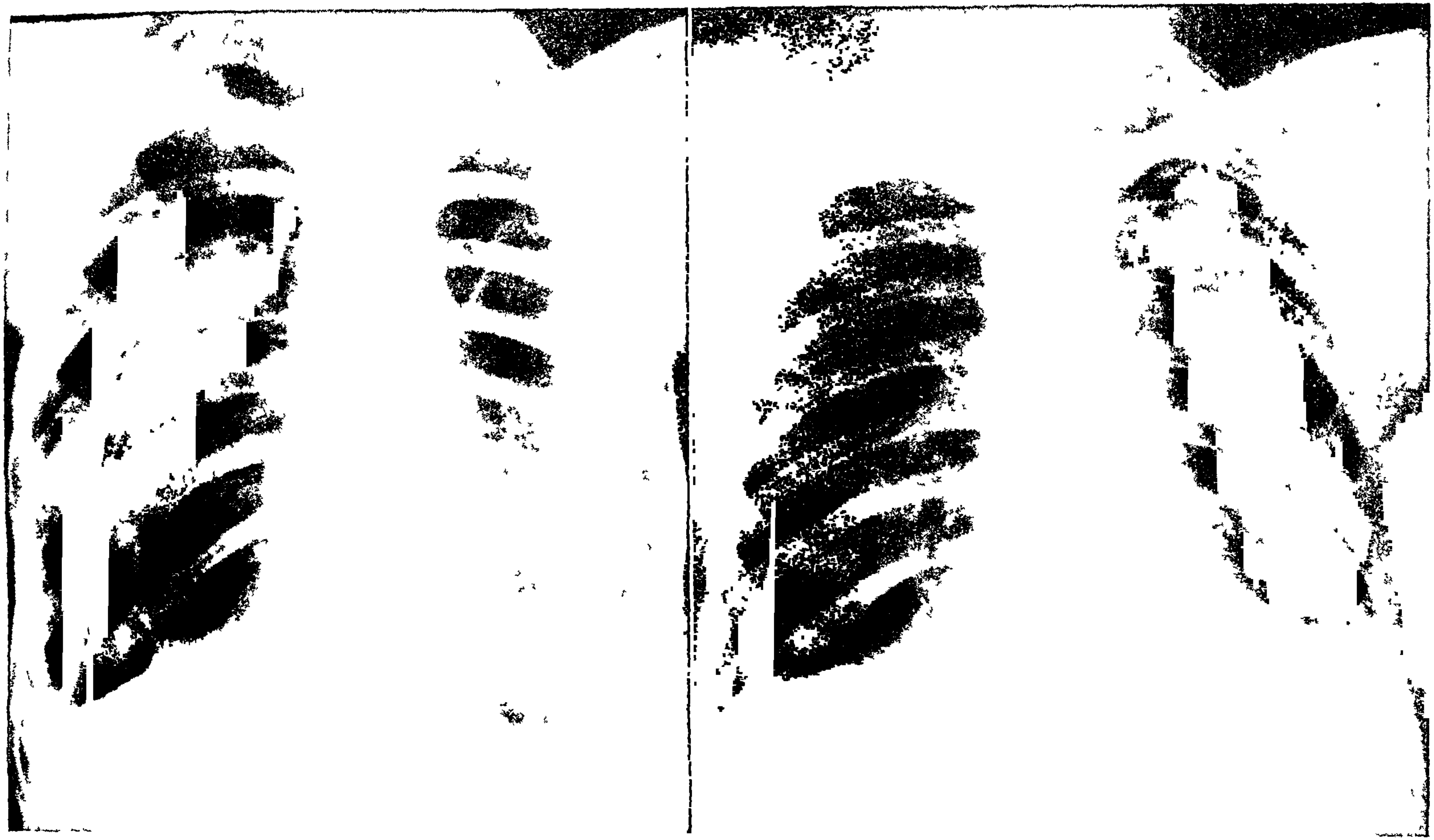
It has been our feeling that the indications for decortication are far wider than the three already enumerated of empyema, hemothorax, and unexpandable pneumothorax lung with persistent space. Essentially, of course, the basic indication is an imprisoned lung which can be expanded either to restore function and/or to obliterate pleural space if such remains. We have grouped the disease conditions indicating decortication in three categories—tuberculous, nontuberculous, and malignant.

TABLE II. INDICATIONS—TUBERCULOUS
115 CASES

| | | |
|---|---|---|
| *Unexpandable pneumothorax lung* | | 39 |
| Unilateral—with space | 30 | |
| Unilateral—with hemorrhage into space | 2 | |
| Bilateral—with space | 2 | |
| Unilateral—without space<br>("False re expansion") | 5 | |
| Restricted lobe(s) limiting expansion at resection | | 55 |
| with expanded pneumothorax | 12 | |
| with "false re expansion" | 6 | |
| without previous pneumothorax | 37 | |
| Restricted lobe at thoracoplasty for postresection residual space | | 8 |
| Tuberculous effusion | | 6 |
| Tuberculous empyema | | 7 |
| with bronchopleural fistula | 5 | |
| without bronchopleural fistula | 2 | |

*Tuberculous Disease.*—Table II lists the indications for decortication in tuberculous disease in our 115 cases. In unexpandable pneumothorax lung, the results have been most gratifying. Thirty-nine patients in our series had previous pneumothorax, 30 having the classical indication of unilateral unexpandable lung with persistent pleural space. Two of these had hemorrhage into the space which remained sterile, and essentially had their procedures done as emergencies because of exsanguination.

Two patients had unexpandable lung on both sides. One of these was so short of breath that he was unable to go to the bathroom without assistance.



A

B

Fig. 1.—Case W. R., a 28-year-old white man. *A*, Preoperative x-ray. *B*, Postoperative x-ray following bilateral decortication.

After decortication of one side he became ambulatory and, after the second decortication, he returned to full activity of strenuous character. This patient was a 28 year old white man who had pneumothorax on the left side from March 1949 until decortication on Nov. 14, 1951, and pneumothorax on the right from March, 1950, until decortication and resection of the posterior segment of the right upper lobe and a wedge resection of a small portion of the anterior segment on March 18, 1953. His pre- and postoperative x-rays are shown in Fig. 1.

So-called "false re-expansion" was a not uncommon occurrence in pneumothorax cases. The presence of a peel prevented the re-expansion of the lung and the space was obliterated by shift of the mediastinum, elevation and fixation of the diaphragm, and contraction of the chest wall. Function was seriously impaired. In each case in this category subjected to operation the condition has proved reversible to an appreciable extent in spite of its persistence for a number of years. Bronchospirometric data, given later, have corroborated this impression. The average duration of "false re-expansion" in our series has been four and one-half years, the longest being seven years.

The largest single group in our series is that of a restricted lobe or lobes limiting expansion at resection. Fifty-five cases have fallen into this category, 12 of the patients having had previous expanded pneumothorax with residual peel restricting the lung, 6 "false re-expansion," and 37 no history of pneumothorax. In the latter, the peel was encountered at surgery and was often unsuspected. In many of the resection cases decortication was of course a secondary procedure. It is a cardinal principle that obliteration of residual pleural space following resection should take place as quickly as possible. Obviously a restricted lobe cannot do this.

When pleural space has persisted after resection and thoracoplasty has been indicated, we have found that decortication of the underlying lung frequently reduces the amount of thoracoplasty necessary.

Tuberculous effusion may result in marked constriction and infolding of the lung on the affected side, with fixation and loss of function of the diaphragm. Decortication can correct this situation. Six of our cases fall into this group. Tuberculous empyema, either with or without bronchopleural fistula, is admittedly more rare than heretofore. We have had seven instances, 5 with fistula and 2 without, in which decortication has been done. Such decortications are more difficult, as there may be no satisfactory line of cleavage between the visceral pleura and the peel. The closure of the bronchopleural fistula frequently results if the space can be obliterated promptly.

*Nontuberculous Disease.*—The conditions for which decortication was performed in the nontuberculous group are given in Table III.

Hemothorax cannot always be managed by aspiration and by enzymatic débridement. Of the 21 cases in our series, 17 were traumatic, 3 spontaneous, and one postoperative. Three of the traumatic patients had a thick calcified plaque restricting the lung following gunshot wounds fourteen, nineteen, and twenty-two years before. In each instance the lung was found to be readily expansile following the removal of the plaque.

TABLE III. INDICATIONS—NONTUBERCULOUS
47 CASES

| | | |
|---|---|---|
| Hemothorax | | 21 |
| traumatic | 14 | |
| spontaneous | 3 | |
| calcified | 3 | |
| postoperative | 1 | |
| Chronic empyema | | 4 |
| Chronic empyema with calcification | | 2 |
| Empyema following resection | | 4 |
| Empyema following ruptured lung abscess | | 2 |
| Spontaneous pneumothorax | | 7 |
| Bronchiectasis | | 3 |
| Bullous emphysema | | 1 |
| Partial agenesis of lung | | 1 |
| Diaphragmatic hernia | | 1 |
| Chylothorax | | 1 |

It is of particular interest that the patient in whom the calcified plaque was present for twenty-two years also had an unsuspected tumor encountered at operation. The presence of the bronchogenic carcinoma was completely masked by the heavy plate of calcium.

Chronic empyema is becoming considerably less common with the lowered incidence of acute empyema and the better handling of cases by thoracic surgeons. Four in our series fell into this category. Two had long-standing empyema with a calcified plaque that had to be removed to permit expansion of the underlying lung. In 4 patients, the empyema followed resection and, in two instances, ruptured lung abscess.

A peel can form rather rapidly if spontaneous pneumothorax persists, particularly in the presence of fluid. Seven patients in our series required decortication when other methods of expanding the pneumothorax were ineffective. In three instances of resection for bronchiectasis, a peel was encountered over nonresected areas that prevented adequate re-expansion. The same was true in one case of bullous emphysema. In the case of partial agenesis of the lung, a thick fibrous peel covered the entire remaining lung tissue. The middle lobe consisted only of a bronchiectatic nubbin which was resected. A peel was also encountered in 3 patients with diaphragmatic hernia, evidently due to inflammatory reaction around the sac. It was necessary to free the lung to restore anatomic relationship. In the case of chylothorax, a lymphoma had eroded the thoracic duct. At exploration a thick peel was encountered.

*Malignant Disease.*—Table IV lists the 10 cases of malignancy for which decortication was performed. The first of these was done in 1945 for uncontrollable pleural effusion due to metastatic malignancy. Obliteration of the space and control of the fluid resulted. In the other 4 similar cases, the same

TABLE IV. INDICATIONS—MALIGNANT
10 CASES

| | |
|---|---|
| Uncontrollable pleural effusion secondary to metastatic malignancy | 5 |
| Restricted lobe(s) secondary to bronchogenic carcinoma | 5 |

result was obtained. We would not imply that decortication should be the procedure of choice in all cases of malignant effusion. Routinely we do pleural biopsy now on all cases of effusion in which the diagnosis has not been established. However, when intrapleural nitrogen mustard and intrapleural isotopes have been ineffective, we have been able by decortication to obliterate the pleural space and obviate the necessity for frequent repeated thoracenteses.

In 5 patients, a peel has been encountered on exploration for bronchogenic carcinoma. In 2 cases, lobectomy was done electively because of low pulmonary reserve, the remaining lobe or lobes being decorticated to fill the space. In the other three instances, a peel was encountered and decortication done of necessity to permit further exploration before inoperability was determined. Needless to say, such a condition is not an indication for decortication in itself.

## SURGICAL TECHNIQUE

Although it is beyond the scope of this paper to describe the operative technique in detail, it is pertinent to enumerate certain surgical maneuvers which we feel are important in preserving and restoring pulmonary function, and which would aid in the interpretation of the complications, the mortality, and the results given in this paper.

In our hands an intercostal incision has been the most satisfactory and has been used in practically all cases. We believe that more satisfactory closure can be obtained, that less postoperative pain occurs, and that better chest wall function results.

Removal of the parietal pleura as well as the visceral peel is always done if sufficient parietal pachypleuritis is present to interfere with return of chest wall motion.

The diaphragm is mobilized to its origin and decorticated, if possible, to permit maximum return of excursion.

All adhesions are freed, fissures developed, and infolding of the lung corrected by removal of overlying webs of peel.

The phrenic nerve must obviously not be injured in the decortication of the mediastinum.

To prevent prolapse, the lung is tacked into optimal position with several catgut sutures, usually at the apex and often at the fissures, before closing the chest. Especially important where raw surfaces are present is the tacking down of the edge of the lower lobe to the diaphragm in the costophrenic sulcus to interpose lung tissue between the surfaces of the diaphragm and the chest wall so that the sulcus will not become obliterated and the pistonlike action of the diaphragm impaired.

Air leaks should be avoided or closed insofar as possible so that maximum prompt re-expansion of the lung can be achieved. Adequate active suction, with replacement of drainage tubes if necesary, is of extreme importance.

Selective breathing exercises, begun prior to operation and continued vigorously postoperatively, even for several months, can do much to restore normal chest wall expansion on the operated side.

It is obvious that a good final functional result must depend greatly upon the technical success of the surgical procedure.

## COMPLICATIONS

Twenty-five complications were encountered in the 172 operated cases. Twelve of these occurred in patients with concomitant pulmonary resection, as shown in Table V. Of these 87 cases, 6 had persistent pleural space requiring the insertion of additional tubes before such space was obliterated. Empyema with bronchopleural fistula resulted in 5 cases. It should be pointed out that these complications were essentially those of the resection and not of the decortication. The same can be said for the tuberculous spread that occurred in the other additional case. This was in a patient undergoing resection under thoracoplasty.

TABLE V. COMPLICATIONS
87 CASES

| DECORTICATION WITH RESECTION | |
|---|---|
| Persistent pleural space | 6 |
| Empyema and bronchopleural fistula | 5 |
| Tuberculous spread | 1 |
| Total | 12 (13.8%) |

The other thirteen complications were found in the 85 cases in which no resection was combined with the decortication. These are listed in Table VI. There were two persistent air leaks necessitating reinsertion of tubes. Two patients had empyema without demonstrable fistula, both being handled without surgical intervention.

TABLE VI. COMPLICATIONS
85 CASES

| DECORTICATION WITHOUT RESECTION | |
|---|---|
| Persistent air leak | 2 |
| Empyema without apparent fistula | 2 |
| Postoperative hemorrhage | 2 |
| Subcutaneous emphysema, severe | 2 |
| Phrenic nerve injury | 2 |
| Persistent pleural space | 1 |
| Chest wall tuberculosis | 1 |
| Tear of superior vena cava | 1 |
| Total | 13 (15.3%) |

Serious postoperative hemorrhage occurred in 2 patients. One, not in our own series, had considerable disease in the underlying lung so that space obliteration was not achieved. Hemorrhage into the space was uncontrolled and death resulted. In the other instance, a thick calcified peel present fourteen years had been removed with some difficulty. Re-exploration of the patient to control the bleeding was necessary. Two patients had severe subcutaneous emphysema requiring superficial skin incisions.

Another complication was that of an inadvertent permanent injury to the phrenic nerve in decorticating the mediastinum. This also occurred in a second patient, but end-to-end anastomosis of the nerve was successful and diaphragmatic function resulted.

One pleural space persisted at the apex; a small thoracoplasty was necessary. Chest wall tuberculosis resulted after decortication in a previously unsuspected tuberculous empyema with routine negative cultures. One of the other fatalities of the series resulted from a tear of the superior vena cava in removing an extremely thick peel.

The cases of hemorrhage and the permanent phrenic paralysis were the only serious complications of this group.

The incidence of complications was 13.8 per cent in the cases of resection with decortication, and 15.3 per cent in the rest of the series.

### MORTALITY

There were four deaths, giving a mortality of 2.3 per cent, in the 172 cases listed in Table VII. The case of operative hemorrhage was the tear of the superior vena cava mentioned under "Complications." The postoperative hemorrhage, also described under "Complications," was in the case of bilateral unexpandable pneumothorax in which decortication was unsuccessful because of severe underlying disease, and hemorrhage could not be satisfactorily controlled.

The other two deaths resulting from pulmonary insufficiency were salvage cases having extremely poor over-all pulmonary function tests prior to surgery. They represent poor operative selection of patients.

TABLE VII. MORTALITY
172 CASES

| | |
|---|---|
| Operative hemorrhage | 1 |
| Postoperative hemorrhage | 1 |
| Pulmonary insufficiency | 2 |
| Total | 4 (2.3%) |

### CLINICAL RESULTS

The results of the 139 cases in whom adequate follow-up was obtained are shown in Table VIII. The criteria by which the patients were classified were both subjective and objective. The great majority of the patients reported that they were able to breathe much better following the procedure than before. They also reported that their exercise tolerance was greater.

The patients were also evaluated as to whether they were incapacitated before and able to return to full activity after the decortication. At physical examination particular attention was paid to the quality of the breath sounds,

TABLE VIII. CLINICAL RESULTS
139 CASES (WITH ADEQUATE FOLLOW-UP)

| | GOOD | FAIR | POOR |
|---|---|---|---|
| Decortication without resection | 50 | 12 | 7 |
| Decortication with resection | 58 | 8 | 4 |
| Total | 108 (77.7%) | 20 (14.4%) | 11 (7.9%) |

TABLE IX. BRONCHOSPIROMETRY

| CASE | DIAGNOSIS | OPERATION | TIMING OF STUDIES | RESTING VENTILATION OPERATED SIDE | VITAL CAPACITY OPERATED SIDE | $O_2$ UPTAKE OPERATED SIDE | VENTILATORY EQUIVALENT $\frac{\text{VENTIL. IN LITERS}}{O_2 \text{ UPTAKE IN C.C.}} \times 100$ | |
|---|---|---|---|---|---|---|---|---|
| | | | | % OF TOTAL | % OF TOTAL | % OF TOTAL | OPERATED SIDE | WELL SIDE |
| 1. R. P. | Pulm. tbc. MAC*<br>Pneumothorax, lt.<br>"False re-expansion" | Pulm. decort., left | Preop. | 15 | 4 | 9 | 2.5 | 1.3 |
| | | | Postop.<br>9 mo. | 40 | 25 | 40 | 1.3 | 1.3 |
| 2. H. C. | Pulm. tbc. MAC<br>Pneumothorax, rt.<br>"False re-expansion" | Pulm. decort., right; wedge, RUL | Preop. | 26 | 17 | 19 | 6.3 | 4.4 |
| | | | Postop.<br>25 mo. | 40 | 34 | 52 | 2.0 | 3.2 |
| 3. E. V. | Pulm. tbc. FAC†<br>Pneumothorax, lt.<br>"False re-expansion" | Pulm. decort., left (previous plombage right) | Preop. | 35 | 27 | 23 | 3.0 | 1.7 |
| | | | Postop. (left)<br>8 mo. | 44 | 44 | 41 | 1.1 | 1.0 |
| 4. A. H. | Pulm. tbc. MAC<br>Pneumothorax, rt.<br>"False re-expansion" | Pulm. decort., right; seg. RUL | Preop. | 47 | 31 | 43 | 1.2 | 1.0 |
| | | | Postop.<br>8 mo. | 51 | 40 | 54 | 2.1 | 2.3 |
| 5. L. M. | Pulm. tbc. FAC<br>Pneumothorax, lt.<br>"False re-expansion" | Pulm. decort., left; seg. LUL | Preop. | 32 | 16 | ? (air leak) | ? | ? |
| | | | Postop.<br>19 mo. | 36 | 33 | 43 | 1.3 | 1.7 |
| 6. D. B. | Pulm. tbc. MAC<br>Pneumothorax space | Pulm. decort., left; seg. LUL | Preop. | 25 | 18 | 20 | 2.5 | 1.9 |
| | | | Postop.<br>36 mo. | 26 | 27 | 33 | 1.0 | 1.4 |
| 7. C. B. | Pulm. tbc. MAC<br>Pneumothorax, lt.<br>Re-expanded | Pulm. decort., left; wedge LUL | Preop. | 41 | 35 | 30 | 2.6 | 1.6 |
| | | | Postop. | 49 | 41 | 36 | 2.4 | 1.4 |
| Mean | | | Preop. | 31.6 | 21.1 | 24.0 | 3.0 | 2.0 |
| | | | Postop. | 40.9 | 42.0 | 42.5 | 1.6 | 1.7 |

*MAC—Moderately advanced, chronic.

†FAC—Far advanced, chronic.

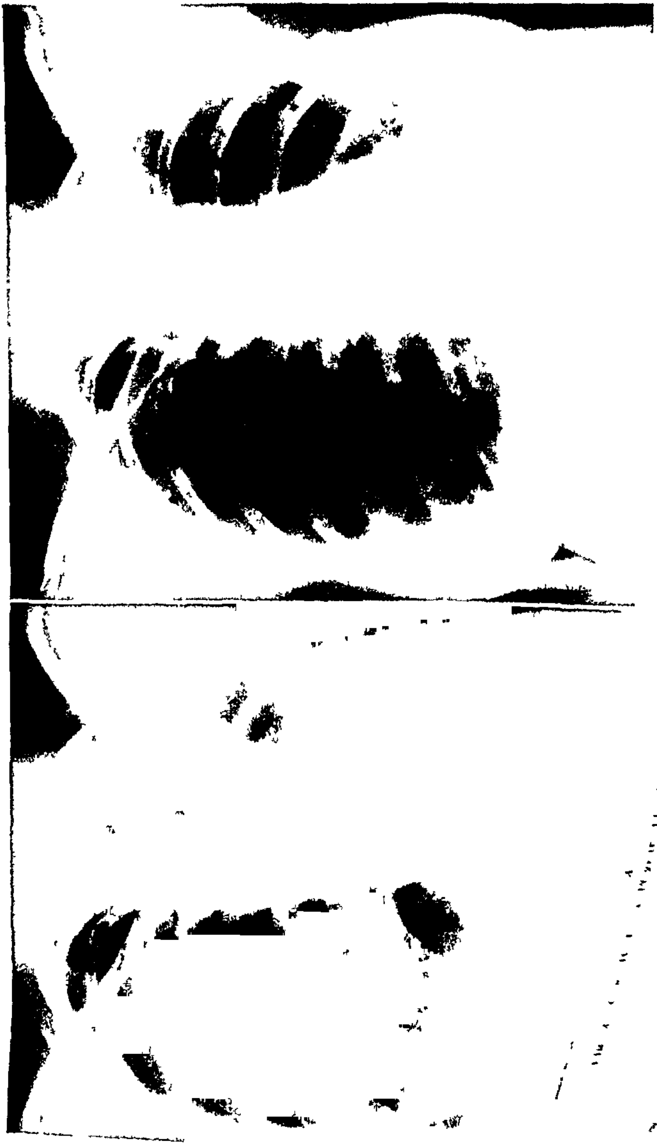


Fig. 4.—Case [illegible] white woman. A, Preoperative x-ray. B, Postoperative x-ray following left decortication.

and their intensification on deep breathing. The extent to which motion of the chest wall returned was noted, as well as the widening of contracted interspaces and the correction of scoliosis. X-ray and fluoroscopy revealed whether the costophrenic angles were satisfactory and whether the excursion of the diaphragm was adequate.

On the basis of these studies, the results were classed as good, fair, or poor. Fifty of the cases in which decortication alone or decortication plus some procedure other than resection was done were classed as a good result, 12 as a fair result, and 7 as a poor result. Four of these poor results were the cases reported under "Mortality." Another was a decortication for metastatic malignancy in which a small empyema persisted.

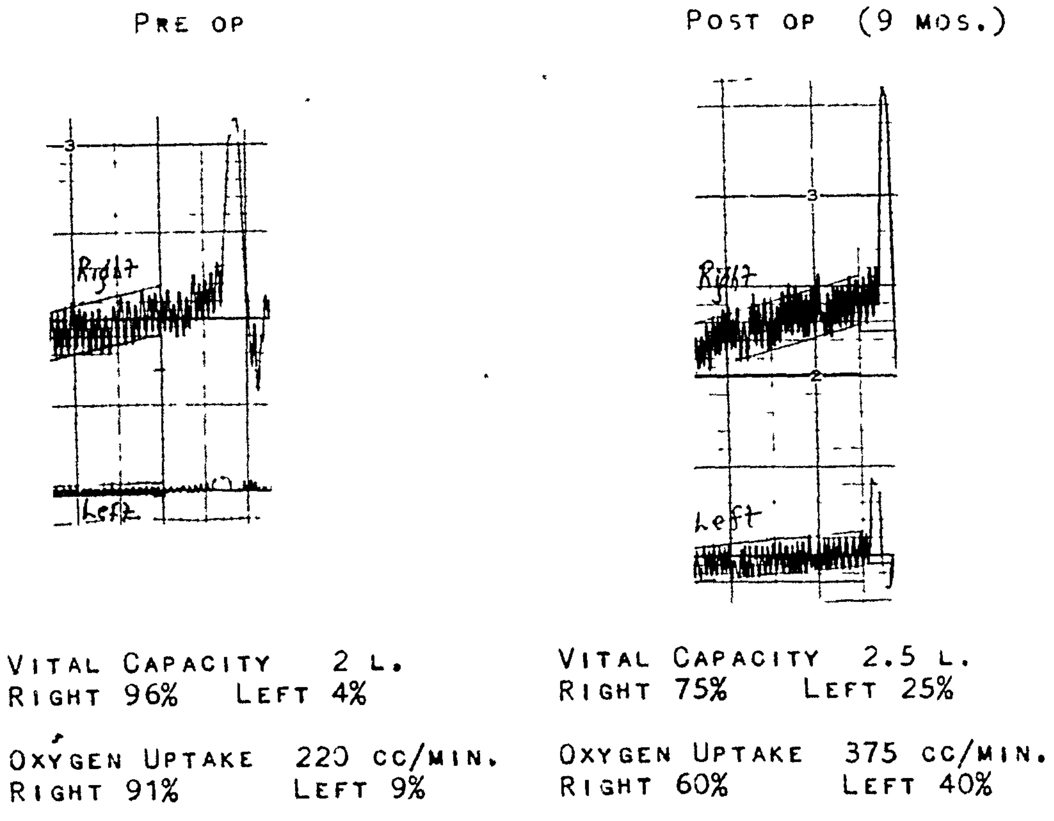


Fig 3.—Case R. P., a 39-year-old white woman. Preoperative and postoperative bronchospirometric tracings and data.

Fifty-eight of the patients having a decortication plus a resection were classed as good, 8 as fair, and 4 as poor.

Of the 139 cases followed up, 108 or 77.7 per cent had a good result, 20 or 14.4 per cent a fair result, and 11 or 7.9 per cent a bad result.

*Pulmonary Function Study Data.*—Although the subject of this paper is the clinical evaluation of pulmonary decortication, the corroborative value of pulmonary function studies is assumed on the basis of the work of Wright,[10] Patton, Watson and Gaensler[11] and others. When feasible, we have done overall ventilatory pulmonary function and bronchospirometric studies pre- and

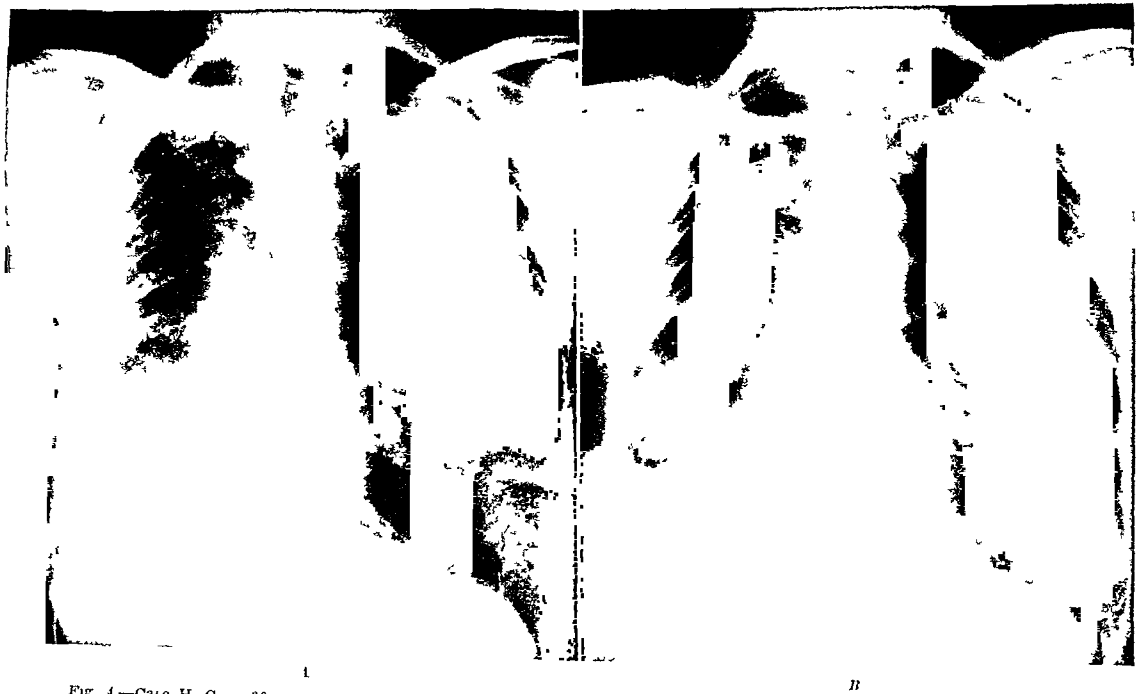


Fig 4—Case H C a 30 year old white man A Preoperative x ray B Postoperative x ray following right decortication

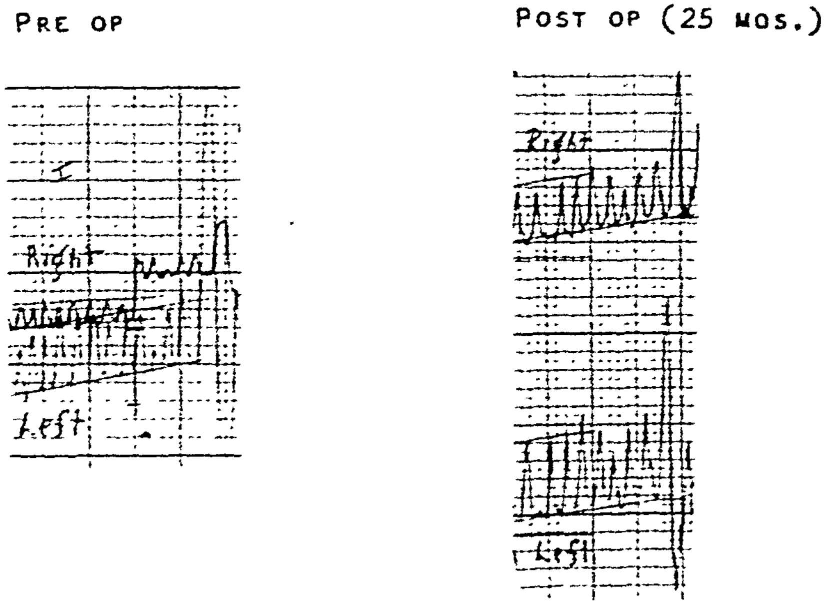
postoperatively. Seven such cases, all tuberculous, having technically satisfactory tracings are presented. Five of these fall into the so-called "false re-expansion" group in which we are particularly interested.

The results show significant improvement by the operated side in every category, that is, resting ventilation, vital capacity, oxygen uptake, and ventilatory equivalent.

Two cases are presented for illustration.

## CASE REPORTS

CASE R. P.—A 39-year-old white woman. Pulmonary tuberculosis, moderately advanced (small cavity LUL). Pneumothorax, left, three years, with "false re-expansion" for four years. Phrenemphraxis, left, regenerated.



Vital Capacity 2.4 L.
Right 17% Left 83%

Oxygen Uptake 210 cc/min.
Right 19% Left 81%

Vital Capacity 2.4 L.
Right 34% Left 66%

Oxygen Uptake 290 cc/min.
Right 52% Left 48%

Fig. 5.—Case H. C., a 30-year-old white man. Preoperative and postoperative bronchospirometric tracings and data.

*Operation:* Pulmonary decortication, left. Fig. 2 shows the pre- and postoperative x-rays, and Fig. 3 the pre- and postoperative bronchospirometric tracings and data.

*Comment:* There was significant increase in ventilation and oxygen uptake on both sides, relatively greater on operated side, following decortication. It is of interest that vital capacity and maximum breathing capacity are both 91 per cent of estimated normal, one year postoperatively.

CASE H. C.—A 30-year-old white man. Pulmonary tuberculosis, moderately advanced (small cavity RUL). Pneumothorax, right, three and one-half years with effusion and "false re-expansion" for two and one-half years.

*Operation* Pulmonary decortication right, plus wedge resection nodule right upper lobe, posterior segment The pre and postoperative x rays are shown in Fig 4, and the bronchospirometric tracings and data in Fig 5

*Comment* There was relative increase in ventilation on operated side, and over all increase in oxygen uptake with relatively greater increase on operated side, following decortication

## SUMMARY

1 A series of 172 cases of pulmonary decortication is presented Seventy patients had decortication alone, 87 decortication with resection, 10 with thoracoplasty, 3 with diaphragmatic herniorrhaphy, and 2 with plication of the diaphragm

2 The disease conditions indicating decortication in tuberculosis included unexpandable pneumothorax lung, either with or without space Eleven cases of "false re expansion" are of particular interest Rehabilitation by decortication of a case of bilateral unexpandable lung is presented The largest group operated upon was composed of those with a restricted lobe or lobes limiting expansion at resection A limited number of restricted lobes found at thoracoplasty were also decorticated Tuberculous effusion and tuberculous empyema were additional indications

3 In nontuberculous disease, hemothorax, either traumatic, spontaneous, or postoperative, was a frequent indication The presence of calcium in the organized hemothorax is not a contraindication Three such cases are reported of fourteen, nineteen, and twenty two years' duration Chronic empyema, with or without calcification, and empyema following resection or lung abscess, indicated decortication in a number of cases in the series Additional cases of spontaneous pneumothorax, bronchiectasis, bullous emphysema, partial agenesis of the lung, diaphragmatic hernia, and chylothorax are enumerated

4 Malignant indications were found where pleural effusion was uncontrollable or peel prevented the expansion of the remaining lobes following elective lobectomy in bronchogenic carcinoma

5 The incidence of complications was 13.8 per cent in the cases in which decortication was combined with resection, and 15.3 per cent in cases without resection

6 There were four deaths in the series, one from operative hemorrhage, one from postoperative hemorrhage, and two from pulmonary insufficiency

7 One hundred and thirty-nine cases with adequate follow-up were evaluated as to results Seventy seven and seven tenths per cent had good results, 14.4 per cent fair results, and 7.9 per cent poor results Deaths were included in the poor results

8 Bronchospirometric studies giving technically satisfactory tracings on a group of 7 tuberculous patients, 5 falling into the group of "false re expansion," are presented Significant improvement was shown by the operated side in resting ventilation, vital capacity, oxygen uptake, and ventilatory equivalent

## CONCLUSIONS

1. Decortication can be applied widely in a number of conditions, tuberculous, nontuberculous, or malignant, stemming from imprisoned lung either with or without a pleural space. Mortality and complication rates are acceptable.

2. "False re-expansion" of a lung following pneumothorax is a valid indication for decortication, as in intractable malignant effusion.

3. The final results of decortication depend not upon the disease for which decortication was done, but upon the two factors of the functioning potential of the underlying imprisoned lung, and the technical success of the surgical procedure.

The cooperation given by Dr. Perry M. Huggin, Medical Director, and Dr. Charles L. Butler, Pathologist, of the East Tennessee Tuberculosis Hospital is gratefully acknowledged.

## REFERENCES

1. Fowler, G. R.: A Case of Thoracoplasty for the Removal of a Large Cicatricial Fibrous Growth From the Interior of the Chest, the Result of an Old Empyema, M. Rec. 44: 838, 1893.
2. Delorme, E.: Nouveau traitement des empyemes chroniques, Gaz. d. hôp., Par. 67: 94, 1894.
3. Delorme, E.: Du traitement des empyemes chroniques par la decortication du Poumon 10 Ieme Congres Français de Chirurgie, p. 379, 1896.
4. Lilienthal, H.: Empyema, Ann. Surg. 62: 309, 1915.
5. Eggers, C.: Radical Operation for Chronic Empyema, Ann. Surg. 77: 327 and 375, 1923.
6. Churchill, E. D.: The Surgical Management of the Wounded in the Mediterranean Theatre at the Time of the Fall of Rome, Ann. Surg. 120: 268, 1944.
7. Burford, T. H., Parker, E. F., and Samson, P. C.: Early Pulmonary Decortication in the Treatment of Posttraumatic Empyema, Ann. Surg. 122: 163, 1945.
8. Mulvihill, D. A., and Klopstock, R.: Decortication of the Nonexpandable Postpneumothorax Tuberculous Lung, J. THORACIC SURG. 17: 723, 1948.
9. Waterman, D. H., and Domm, S. E.: Decortication of the Unexpandable Pneumothorax Lung, Dis. Chest 19: 1, 1951.
10. Wright, G. W., Yee, L. B., Filley, G. F., and Stranahan, A.: Physiologic Observations Concerning Decortication of the Lung, J. THORACIC SURG. 18: 372, 1949.
11. Patton, W. E., Watson, T. R., Jr., and Gaensler, E. A.: Pulmonary Function Before and at Intervals After Surgical Decortication of the Lung, Surg., Gynec. & Obst. 95: 477, 1952.

## DISCUSSION

DR. ALLAN STRANAHAN, Albany, N. Y.—We too have been interested in pulmonary function studies before and after decortication. I would like to present one case which illustrates the value of preoperative bronchograms in deciding whether or not one should do a resection with a decortication.

(slide) This 60-year-old white man's present illness began in 1910 when he had pneumonia followed by an empyema on the left. At that time the left pleural cavity was drained and bismuth paste was instilled in the pleural space at the time of drainage. He gradually regained his stamina and was apparently well until 1950 when he again had symptoms of sepsis, chills and fever, night sweats, increased shortness of breath, with cough productive of large amounts of thick yellow sputum. The left pleural cavity was tapped and *Staphylococcus aureus* was obtained in culture.

(slide) A bronchogram was made to identify the extent of pulmonary disease and this showed fairly complete filling of the smaller branch bronchi of the upper lobe with rather marked distortion and contraction of the lower lobe segments. These findings indicate pleural parenchymal fibrosis with scarring of the lung, and under ordinary circumstances are an indication for pulmonary resection in conjunction with decortication.

(slide) The poor pulmonary function studies and the patient's history of dyspnea indicated that he was a severe cardiopulmonary cripple. The maximum breathing capacity was only 37 L. per minute. The lung volume studies showed a high residual air, indicating considerable trapping of air and probable irreversible pulmonary emphysema. The bronchospirometry showed a poorly functioning left lung with some discrepancy of oxygen consumption and diminished ventilation. Accordingly, it was felt that the operation of choice in this particular patient should be that of decortication alone, rather than decortication and pulmonary resection, recognizing the fact that we would be leaving residual pulmonary disease.

(slide) Decortication was done, and this slide shows the bismuth paste which had been in the pleural cavity for forty years and also the cortical peel which was removed.

(slide) The postoperative result was not dramatic. However, the infection was removed and the upper lobe following decortication expanded quite well. There were multiple small bronchial fistulas arising in the superior segment of the lower lobe which were simply closed with fine catgut. The patient was discharged from the hospital without complications and at the present time is doing well.

(slide) The postoperative pulmonary function studies showed a slight increase in the maximum breathing capacity. The residual volume decreased a slight amount. There was some improvement in the oxygen consumption and ventilation of the left lung. We did not, however, feel that this helped this patient a great deal from the standpoint of pulmonary function.

There is one other point I would like to make which Dr. Waterman only briefly mentioned, that is the case of decorticating a lung in a patient who develops an empyema following pneumothorax, either therapeutic or spontaneous. In our experience, these lungs are rather easy to decorticate in contradistinction to those in patients whose empyema develops primarily without preceding pneumothorax. This latter type of patient usually has a severe degree of pleuroparenchymal scarring which makes decortication difficult if not impossible. However, if the onset is that of a primary tuberculosis or nontuberculosis empyema then, with the pleuroparenchymal fibrosis that is present, decortication is very difficult.

DR. JOHN S. CHAMBERS, San Diego, Calif.—I enjoyed Dr. Waterman's paper very much and wish to direct the following remarks to his comments about decortication for neoplastic pleural effusion. In patients with pleural effusion, secondary to neoplasm both primary in the lung and metastatic, intercostal intubation and instillation of a suspension of talcum powder has been used with the idea of producing an obliterative pleuritis and obviating frequent chest taps. In 20 of 23 patients no further thoracenteses were necessary.

I believe these results are superior to those obtained with radioactive gold and that this procedure would make decortication for palliation unnecessary in neoplastic effusion.

DR. GABRIEL SELEY, New York, N. Y.—This is a timely subject and I want to thank Dr. Waterman for reintroducing it. I would like to stress the use of decortication in acute and subacute empyemas. We have seen an increasing number of acute empyemas due to resistant strains of microorganisms, especially hemolytic *Staphylococcus aureus*. We as chest surgeons are usually not consulted until after our medical colleagues have already used multiple aspirations and the conventional antibiotics have failed, due to loculations present in the lobar distribution of the empyema, or bacterial resistance to the drugs employed.

I am not talking about infected pleural fluid, but am trying to discuss a typical purulent pleuritis, a real empyema with pus. At this stage, enzymatic decortication is usually ineffective. Simple drainage may and often will result in chronic empyema, whereas a properly conducted decortication will prevent chronic empyema and restore lung function more rapidly and effectively. At the time of operation, one must pay particular attention to all the loculations, especially those beneath the lung and between the lobes. With the liberal use of blood transfusions and properly tested wide spectrum antibiotics, complete decortication can be performed safely even in the extremely sick and toxic patient and, in fact, can be lifesaving. In addition, pulmonary foci, if present, can also be handled at the same time. Multiple tube suction should be instituted and continued for a long period, postoperatively.

A number of recent patients including adults and children treated in this manner have had complete and rapid restitution of normal anatomy and physiology.

DR. R. LEONARD KEMLER, Hartford, Conn.—First, I should like to commend Dr. Waterman for the excellent presentation of a very interesting subject. There was one line in one of the slides which interested me greatly; I believe it stated that he had used decortication for malignant pleural effusions in 5 cases. We have had experience in one case in which a patient came to us with a pleural effusion which contained malignant cells on several occasions; despite repeated tappings the effusion would recur every ten days. The patient was given x-ray therapy, radioactive gold and nitrogen mustard, and the pleural effusion continued to reaccumulate at a very rapid rate. The man was 55 years of age and in desperation. In order to make his remaining days as comfortable as possible, we attempted a decortication. To our surprise we were able quite easily to decorticate the lung, the pleural of which contained multiple tumor nodules. The parietal pleural surface was removed quite easily also. The patient lived for a year and one half afterward with no further tapping and with a very comfortable existence. I would like to hear Dr. Waterman's comments on the 5 cases in which he tried it.

DR. WILLIAM K. ROGERS, Knoxville, Tenn.—I would like to show one case of bilateral pneumothorax in which the lung was decorticated because of failure of re-expansion on both sides.

(slide) This is the x-ray at the time we saw the patient. He was unable to get out of bed to go to the bathroom by himself because he was so short of breath. The left side was attacked first.

(slide) This shows the peel that was removed on the left side.

(slide) This is the postoperative film after the left side had been decorticated.

(slide) This is the peel subsequently removed on the right.

(slide) This is the final film.

After the bilateral procedures had been carried out this individual was rehabilitated considerably. He now carries on one of the most strenuous occupations in eastern Tennessee, that of a Baptist preacher.

I would like to elaborate on one other point. As Dr. Waterman said, the purpose of decortication is twofold; to restore function and to obliterate pleural space. In going over one's cases, one may think he has had a poor result because the x-rays do not look good, or perhaps because the diaphragm does not move quite as well as he might wish. However, one must take into account what he wished to accomplish in the first place. It may not be necessary to have a perfect-looking x-ray; it may not be necessary to have a normally functioning diaphragm. If the patient is rehabilitated and if the pleural space has been closed and infection has been controlled, then decortication is certainly a very worth-while procedure regardless of the appearance of the x-ray.

DR. PAUL C. SAMSON, Lafayette, Calif.—I want to thank Dr. Waterman for reopening this subject. It became rather passé after the war and yet, as you can see, the indications have everwidened and I think it is worth while to reconsider them again. Likewise, I would like to thank him for his kind remarks toward your recorder.

I have two observations to make. The first is in relation to the use of some type of decortication in malignant disease. We have had a brief experience with this and I recall in particular 2 young children who had malignant disease and uncontrollable fluid to the extent that they were being aspirated every third or fourth day, as Dr. Kemler's patient was. Each had had x-ray therapy without success and, in each, a combination of decortication and parietal pleurectomy enabled the lung to re-expand; no further thoracenteses were necessary. Of course both youngsters died of their malignant disease but during the number of months that they lived they did not have to be aspirated.

I would take issue with Dr Waterman on one point. He mentioned that there is no peel on the parietal pleura, if I heard correctly. I would remind you that there is a peel on the parietal pleura and, while the parietal pleura may thicken an infinitesimal amount, still and all we can decorticate the parietal peel and do so very frequently. I will agree, however, that in older and more chronic disease, technically it is much easier to do a pleurectomy than it is to do decortication on the parietal side and therefore I agree inso far as he says pleurectomy is a preferable procedure. But there is a peel on the parietal pleural side, as one can tell if he examines these specimens carefully under the microscope

Thank you very much It was a good paper.

DR. JAMES E DAILEY, Houston, Texas—I merely wish to mention briefly that for quite a few years, in dealing with patients with nonresectable nonmalignant lesions and especially with those having associated pleural effusion, I have made it a habit to perform a parietal pleurectomy almost routinely with very gratifying results The necessity for repeated aspiration stops with re expansion and pleural symphysis and even more gratifying is the fact that these pleurectomized patients remain much more comfortable with respect to pain In only very few cases have I taken the trouble to decorticate the visceral pleura Parietal pleurectomy seems to suffice

DR A. R VALLE, Detroit, Mich—I would like to congratulate Dr. Waterman on this excellent presentation and his good results However, I thoroughly agree with Dr Samson that both the visceral and parietal peels should be removed. I feel that it is very important to remove the parietal peel because it is almost always the thicker and it immobilizes the rib cage and diaphragm, thus interfering with pulmonary function Also, I think it is important to start active physical therapy soon after surgery since it aids in restoring the rib cage and lung to normal function In our series of 278 nontuberculous patients, we obtained 91 per cent good results, 5 per cent fair results, and 4 per cent poor results. I must mention, however, that the majority of the patients in our series were young soldiers wounded in the Korean War, which no doubt influenced our results

DR RICHARD H. MEADE, Grand Rapids, Mich—I was particularly interested in the use of this procedure in malignant disease It seems quite logical that if you do a decortication and remove the parietal pleura, you will open up many channels of absorption. I should like to know what happens in cases where you do not do that About eight months ago, I operated on a patient who had pleural effusion We could not make a definite diagnosis of the source of it At operation I found that she had an inoperable carcinoma of the lung All I did was make a biopsy of the lung tumor There has not been a drop of fluid since that time I do not believe we know just what happens in such cases Those of you who can remember many years ago when tuberculous peritonitis was seen fairly frequently will remember that after exploratory celiotomy frequently the fluid would disappear promptly

DR. DAVID H. WATERMAN (Closing)—I should like to thank the discussants for their remarks and for their approbation We have been particularly impressed by this group of malignancies that Dr. Kemler and several others have mentioned Certainly, we would not wish to imply that decortication should always be done in malignant effusion; obviously not When there is an unexpandable lung with intractable fluid, however, as not infrequently occurs, the operation may be of practical use Our first case, in 1945, was remarkably successful in eliminating the troublesome effusion, and our subsequent cases have also done well, with the exception of one patient who may have had his death speeded by the opening of new channels for malignant spread, as Dr. Meade mentioned

It would be presumptuous of me to take issue with an authority on decortication of Dr Samson's physical and professional stature I was quoting an article that appeared in 1951 in the Journal of this Association by Croxatto and Sampietro, the former a pathologist and the latter a thoracic surgeon at the University of Buenos Aires, who emphatically stated that there was actually a pachypleuritis on the parietal side and not a true peel, and that it was therefore not possible to separate a peel from the parietal pleura We had felt that it was possible, and had always used the term "pari

We suggest that perhaps a peel is present in the very early cases, and a pachypleuritis in the long-standing ones. However, we do think that removal of the parietal peel or parietal pleura, whichever the case may be, is mandatory in instances where there is marked thickening. It is inconceivable to me that a thick shoe-leather peel over the parietal surface will disappear following decortication of the visceral side alone and permit return of good function of the chest wall.

Dr. Sarot's* comment as to the misnomer perhaps is well taken. I doubt if we are going to be able to change the terminology at this late date because it is so well ingrained in the literature.

I would like to mention briefly one factor that he stressed, namely, the condition of the underlying lung. Obviously it is extremely important to evaluate the pre-existing and existing pulmonary disease if a good result is to be obtained. This is particularly true in tuberculosis. If no appreciable disease is present, however, the duration of collapse does not seem to affect the re-expansion. In our cases of calcified hemothorax, for example, one had been collapsed thirteen years, one nineteen years, and the other twenty-two years since the original gunshot wound. Yet in each case the lung decorticated satisfactorily with the use of bone instruments—the plaque had to be literally chiseled and rongeured away, but once it was removed the lung expanded beautifully, with complete space obliteration on the table.

As a point of interest, the case of calcified hemothorax of twenty-two years' duration had an unsuspected bronchogenic cancer in the upper lobe. This was completely masked by the overlying calcium, and it was *not until decortication* that the tumor was found. Unfortunately, it was not resectable. Conceivably we have in such instances another rare indication for decortication.

---

*Dr. Sarot did not return his discussion. For that reason it could not be included here. EDITOR.

# A SURGICAL PATHOLOGIC CLASSIFICATION FOR ISOLATED VENTRICULAR SEPTAL DEFECTS AND FOR THOSE IN FALLOT'S TETRALOGY BASED ON OBSERVATIONS MADE ON 120 PATIENTS DURING REPAIR UNDER DIRECT VISION

HERBERT E. WARDEN, M.D.* (BY INVITATION), RICHARD A. DEWALL, M.D.** (BY INVITATION), MORLEY COHEN, M.D. (BY INVITATION), RICHARD L. VARCO, M.D., AND C. WALTON LILLEHEI, M.D.
MINNEAPOLIS, MINN.

BETWEEN March 26, 1954, and May 5, 1956, at the University of Minnesota Hospitals, 144 patients with intracardiac lesions underwent direct vision repair of their defects while under total cardiac by-pass and were arterially perfused via controlled cross circulation[1-6] (45 patients), the arterial reservoir[7, 8] (5 patients), the biologic (dog lung) oxygenator[9] (14 patients), or the mechanical (bubble) oxygenator[10, 11] (80 patients).

In this series the most common congenital defect encountered has been an isolated ventricular septal defect (87 patients†). Next in frequency was the tetralogy of Fallot defect (33 patients), and then the lesion of atrioventricularis communis (9 patients). These three types comprise 89.5 per cent of the cases operated upon. The first two types of ventricular septal defects will receive our attention in this publication. The atrioventricularis communis defect, although possessing a ventricular component, has in addition an ostium primum defect and, as well, mitral and/or tricuspid valvular malformations. Therefore, in the light of its complexity, it warrants and will receive a separate communication.

The common denominator in all of these patients operated upon was their recurring illness. They were periodically in need of pediatric or medical care. This fact may well have influenced for this series the frequency distribution of the various defects.‡ The acyanotic cases were in difficulty from numerous respiratory infections and/or bouts of cardiac failure. Pulmonary hypertension was universally present here. Among those who were cyanotic, our surgical indications were based on progressive and sustained deterioration as manifested by one or more of the following conditions: a rising hemoglobin and/or

---

From the Department of Surgery and the Variety Club Heart Hospital, University of Minnesota Medical School, Minneapolis, Minn.

Supported by Research Grants from: Graduate School, University of Minnesota; Minnesota Heart Association; American Heart Association; Life Insurance Medical Research Fund; and National Heart Institute, Grant No. H-830.

Read at the Thirty-sixth Annual Meeting of The American Association for Thoracic Surgery at Miami Beach, Fla., May 7 to 9, 1956.

*Research Fellow, United States Public Health Service.

**American Heart Association Research Fellow.

†This includes one case of traumatically acquired ventricular septal defect successfully repaired.[9]

‡The ratio of low septal defects to high ones may be greater than our series shows. The pressing need to care for children in serious trouble from a high ventricular septal defect has effectively precluded scheduling of the more bland forms occasionally met with in older children and adults.

hematocrit (with the emphasis upon the rate at which this ascent was occurring); recurrent severe bouts of dyspnea associated with syncope, coma, or convulsions; and, in some, clearly evident cardiac decompensation.

## CLASSIFICATION OF VENTRICULAR SEPTAL DEFECTS

It is both logical and desirable to classify the diverse types of septal anomalies. Consequently, we have sought a simple yet concise and inclusive classification for ventricular septal malformations as we have found them at operation. One was looked for which would be both morphologically correct and equally compatible with the antecedent embryologic aberrations. Rokitansky's anatomic classification of congenital cardiac septal defects as described in 1875,[12] we believe, best fulfills most of the above requirements. We have found that it offers the surgeon an accurate, workable, anatomic classification which, except for a few minor reservations, is essentially in agreement with the embryologic descriptions of Spitzer[13] and others.[11-16] The portion of this classification that deals with ventricular septal defects in its modified form is reproduced in Table I.

TABLE I. ANATOMIC CLASSIFICATION OF VENTRICULAR SEPTAL DEFECTS (AFTER KARL FREIHERR VON ROKITANSKY* 1804-1878)

A. Complete absence of ventricular septum (single ventricle)
B. Defect in the posterior ventricular septum
C. Defect in the anterior ventricular septum
  1. Absence of the entire anterior septum
    a. With normal caliber of the arterial trunks
    b. With stenosis or atresia of the pulmonary artery (tetralogy of Fallot)
  2. Defect in the posterior part of the anterior septum
    a. With anomalous position of the arterial trunks
      Alpha = With normal caliber of the arterial trunks
      *Beta = With stenosis or atresia of the pulmonary artery (tetralogy of Fallot)*
    b. With normal position of the arterial trunks (membranous type)
  3. Defect in the anterior part of the anterior septum
D. Defect in unusual positions (including muscular defects)
E. Defect in anomalous septum
*F. Acquired ventricular septal defects

*With modifications described in text.

A cross section of the heart at the level of the atrioventricular ring is shown (Fig. 1, *A* and *B*) to facilitate the description and identification of the areas concerned in Rokitansky's classification. The relationship between these structures and the ventricular septum is schematically represented in Figure 1, *C*. The latter is taken from the works of Spitzer[13] and for simplicity has been modified slightly. The stippled areas represent the muscular portions of the ventricular septum while the solid line (*3-5*), joining these regions, represents the pars membranacea. The ventricular septum at this level may be segmented into an anterior or ventral portion (Fig. 1, *C, 1-4*) and a posterior or dorsal part (*4-6*) which corresponds to the embryologic source of these regions.

The anterior septum is derived from a union of the ventral atrioventricular cushion with the muscular ventricular septum below and the right and left conus ridges from above. The posterior ventricular septum arises from the dorsal atrioventricular cushion and the portion of the muscular ventricular septum that arises from the apical and dorsal ventricular walls of the embryo.[14, 15] The central point of fusion of these regions occurs at *1* (Fig. 1, *C*) in the pars membranacea. The anlagen of the constituents of this membranous area of the septum have been a subject of dispute among embryologists for some time.

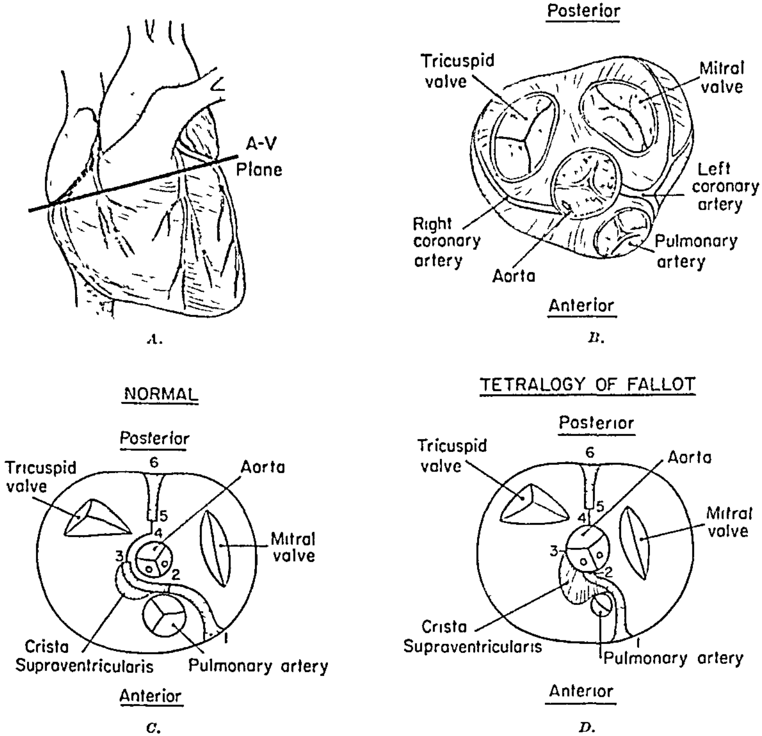


Fig. 1.—The heavy cross bar in *A* indicates the level of the atrial-ventricular junction. *B* depicts the gross anatomy at this level.

The relationships of the mitral and tricuspid valves and the aorta and pulmonary artery with respect to the ventricular septum of the normal heart are schematically indicated in *C*. The stippled areas indicate the muscular portion of the ventricular septum while the heavy line between numbers *3* and *5* indicates the membranous section. In *D*, the pathologic cardiac anatomy found in tetralogy of Fallot is indicated. (See text for details concerning the septal divisions indicated in the latter two plates.)

However, in summary, the present concept holds that the anterior superior portion of the membranous septum arises from connective tissue contributed by the left conus ridge which extends down from the base of the left bulbar ridge while the posterior superior portion comes from the right conus ridge; these segments then join and in turn unite with the fused atrioventricular cushions. The remaining inferior portion of the membranous septum is contributed mainly by the dorsal atrioventricular cushion though some does come from its ventral counterpart and from the connective tissue margin of the crest of the muscular interventricular septum.[15] Thus, it is obvious that this region has a complex embryologic origin and that fact undoubtedly contributes to the frequency of malformations in this area.

These major divisions of the ventricular septum are further subdivided by Spitzer as indicated in Fig. 1, *C*. The anterior ventricular septum is composed of anterior and posterior divisions. The anterior division extends from the anterior ventricular wall (*1*) to a point midway beneath the aorticopulmonic septum (*2*); the posterior portion (*2-4*) is subdivided into a muscular part (*2-3*) and a membranous section (*3-4*) which extends to the anterior margin of the posterior ventricular septum (*4*). This latter structure is made up of a small membranous segment (*4-5*) and a larger muscular section between *5* and *6* which terminates at the dorsal ventricular wall. These subdivisions by Spitzer, based upon embryologic derivation have been correlated with Rokitansky's morphologic classification and the clinical cases have been grouped according to these terms in Table II.

TABLE II. CLASSIFICATION OF THE CLINICAL MATERIAL IN TERMS OF ROKITANSKY'S GROUPS AND SPITZER'S AREAS

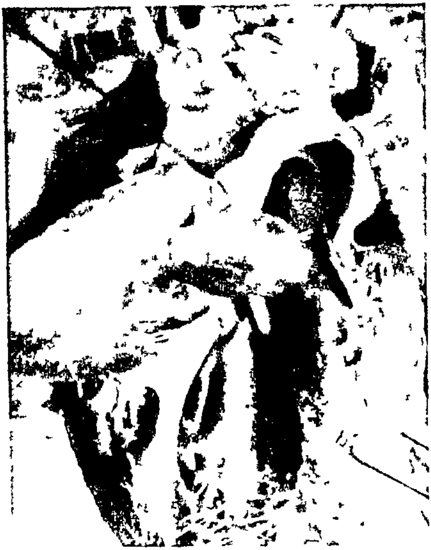
| ROKITANSKY'S ANATOMIC GROUPS | SPITZER'S SEGMENTS | NUMBER OF PATIENTS REPAIRED |
|---|---|---|
| A | 1-6 | -- |
| B | 4-6 | 5 |
| C 1 a | 1-4 | 1 |
| b (with pulmonary stenosis) | 1-4 | 3 |
| C 2 a alpha | 2-4 | 2 |
| beta (with pulmonary stenosis) | 2-4 | 30 |
| C 2 b | 2-4 | 71 |
| C 3 | 1-2 | 4 |
| D | No correlation | 3* |
| E | No correlation | 1 |
| F Traumatic | -- | 1 |
| Total | | 121 |

*One of these also had a C, 2b type defect.

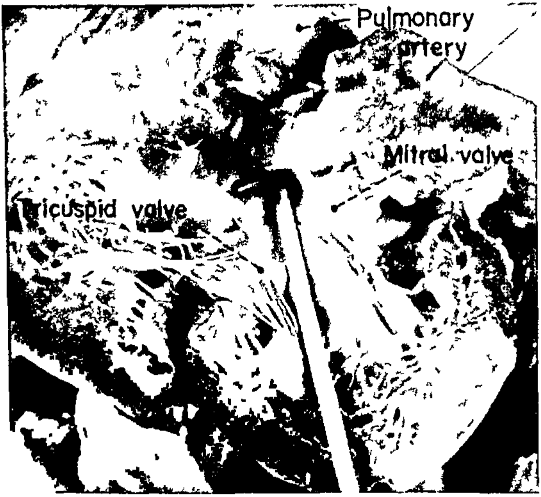
The areas just described and shown in Figure 1, *C* have proved adequate for categorizing the majority of the ventricular septal defects we have observed in this series and those encountered in the tetralogy of Fallot group. The anatomic features of this latter malformation have been diagrammed in Fig. 1, *D* which shows the enlarged aorta, the hypertrophied crista supraventricularis, and the narrowed pulmonary outflow tract. The details of this septal anomaly will be elaborated upon later. The schema here is also modified from the works of Spitzer and the nomenclature is his.

Group A: *Complete Absence of the Ventricular Septum (Single Ventricle).*—

This group is characterized by the complete, or virtually so, failure of the muscular ventricular septum to develop. The atrioventricular valves are at times distorted but empty into a single chamber. In the roof of this common ventricle appear the orifices of the aorta and pulmonary artery. Though these vessels may be normal, frequently they too are imperfectly formed or transposed. Occasionally, the pulmonary artery is markedly atretic or completely absent.



*A*



*B*

Fig 2.—Single ventricle (Rokitansky's Group A). These are photographs of a heart from a 4 month old infant and demonstrate the pathologic anatomy of one type of single ventricle with normal position and caliber of the great vessels. *A* shows the pulmonary outflow tract in the upper right foreground. *B* is a view from the apex toward the roots of the ventricular outflow tracts. In each instance the probe extends out the aorta.

The aorta can be similarly deformed. Fig. 2 (*A* and *B*) are photographs of a representative specimen of one type of single ventricle with normal atrioventricular valves and normal position and caliber of the great vessels. In Fig. 2, *A* the incision in the ventricular cavity has been extended from the apex well out the pulmonary artery and the orifice of the latter structure is in the foreground. A *probe passes out through the aorta. In Fig. 2, B the specimen has* been more widely opened and the base of the great vessels is more apparent as are the tricuspid and mitral valves. The probe again extends out the aorta. As yet we have not undertaken surgical correction for this group of cases.

Group B: *Defect in the Posterior Ventricular Septum.*—

Defects of this portion of the ventricular septum are represented in Fig. 3 (*A* and *B*). It can be noted that here one margin of this defect is in juxtaposition to the atrioventricular ring or annulus, and that it is well concealed by the septal and, to some extent, the posterior leaflets of the tricuspid valve. In addition, because of a close proximity to the atrioventricular ring, the upper margin of such a defect is usually quite fibrous while the lower margin is composed predominantly of muscle.

These lesions may vary from that of a hole but a few millimeters in diameter and juxtaposed to the posterior margin of the membranous septum to an almost total absence of the posterior septum extending from the pars membranacea to the posterior ventricular wall. We have cataloged 5 patients in this series as having this type of defect. In 3 cases the opening appeared smaller than that shown in Fig. 3, *B*. Their range was between 1.5 and 2.5 cm. However, among these smaller holes encountered, adequate surgical exposure may be more troublesome and hence secure closure more difficult.

Group C: *Defect of the Anterior Ventricular Septum.*—

1. *Absence of the entire anterior septum:* A heart with this portion of the septum deficient and with normal caliber of the arterial trunks is illustrated in Fig. 4. The openings found so far have all been large and extended from just beneath the pulmonary valve cusps well back toward the anterior margin of the septal leaflet of the tricuspid valve. The aortic and pulmonary valve leaflets are separated by a narrow (2 to 4 mm.) fibrous band, leaving just enough room for the precise placement of closure stitches.

Four patients in the series had this lesion. In only one was there a normal outflow tract. The others had definite pulmonary outflow stenosis (tetralogy of Fallot). This is an unusual form of tetralogy since the supraventricular crest has appeared to be entirely absent, whereas, it is customarily hypertrophied (C, 2a, beta). The appearance at the time of right ventriculotomy is strikingly different in these two contrasting examples. Rokitansky seemingly did not observe any defects of this portion of the ventricular septum in association with pulmonary stenosis or atresia and hence did not make provision for their inclusion in his classification. Consequently, we have modified his original schema (see Table I) by this addition.

Group C, 2a, alpha: *Defect in the posterior portion of the anterior septum with anomalous position of the arterial trunks which are of normal caliber:* This group includes those septal defects which are associated with anomalies

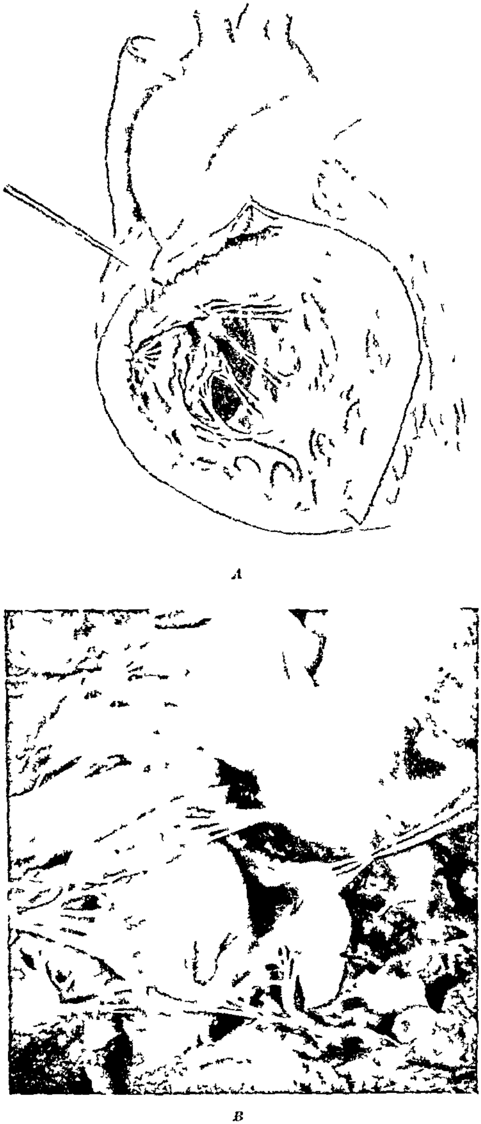


Fig 3.—Defect of the posterior septum (Rokitansky's Group B) *A* is an artist's version of a defect in the posterior ventricular septum and *B* shows a photograph of a similar defect taken from a view similar to that seen through a surgical cardiotomy in the right ventricle In this view the pulmonary valve cusps are visible at the top of the photograph and the manner in which the septal and posterior leaflets of the tricuspid valve tend to hide partially the margins of the defect is quite evident

The aorta can be similarly deformed. Fig. 2 (*A* and *B*) are photographs of a representative specimen of one type of single ventricle with normal atrioventricular valves and normal position and caliber of the great vessels. In Fig. 2, *A* the incision in the ventricular cavity has been extended from the apex well out the pulmonary artery and the orifice of the latter structure is in the foreground. A probe passes out through the aorta. In Fig. 2, *B* the specimen has been more widely opened and the base of the great vessels is more apparent as are the tricuspid and mitral valves. The probe again extends out the aorta. As yet we have not undertaken surgical correction for this group of cases.

Group B: *Defect in the Posterior Ventricular Septum.*—

Defects of this portion of the ventricular septum are represented in Fig. 3 (*A* and *B*). It can be noted that here one margin of this defect is in juxtaposition to the atrioventricular ring or annulus, and that it is well concealed by the septal and, to some extent, the posterior leaflets of the tricuspid valve. In addition, because of a close proximity to the atrioventricular ring, the upper margin of such a defect is usually quite fibrous while the lower margin is composed predominantly of muscle.

These lesions may vary from that of a hole but a few millimeters in diameter and juxtaposed to the posterior margin of the membranous septum to an almost total absence of the posterior septum extending from the pars membranacea to the posterior ventricular wall. We have cataloged 5 patients in this series as having this type of defect. In 3 cases the opening appeared smaller than that shown in Fig. 3, *B*. Their range was between 1.5 and 2.5 cm. However, among these smaller holes encountered, adequate surgical exposure may be more troublesome and hence secure closure more difficult.

Group C: *Defect of the Anterior Ventricular Septum.*—

1. *Absence of the entire anterior septum:* A heart with this portion of the septum deficient and with normal caliber of the arterial trunks is illustrated in Fig. 4. The openings found so far have all been large and extended from just beneath the pulmonary valve cusps well back toward the anterior margin of the septal leaflet of the tricuspid valve. The aortic and pulmonary valve leaflets are separated by a narrow (2 to 4 mm.) fibrous band, leaving just enough room for the precise placement of closure stitches.

Four patients in the series had this lesion. In only one was there a normal outflow tract. The others had definite pulmonary outflow stenosis (tetralogy of Fallot). This is an unusual form of tetralogy since the supraventricular crest has appeared to be entirely absent, whereas, it is customarily hypertrophied (C, 2a, beta). The appearance at the time of right ventriculotomy is strikingly different in these two contrasting examples. Rokitansky seemingly did not observe any defects of this portion of the ventricular septum in association with pulmonary stenosis or atresia and hence did not make provision for their inclusion in his classification. Consequently, we have modified his original schema (see Table I) by this addition.

Group C, 2a, alpha: *Defect in the posterior portion of the anterior septum with anomalous position of the arterial trunks which are of normal caliber:* This group includes those septal defects which are associated with anomalies

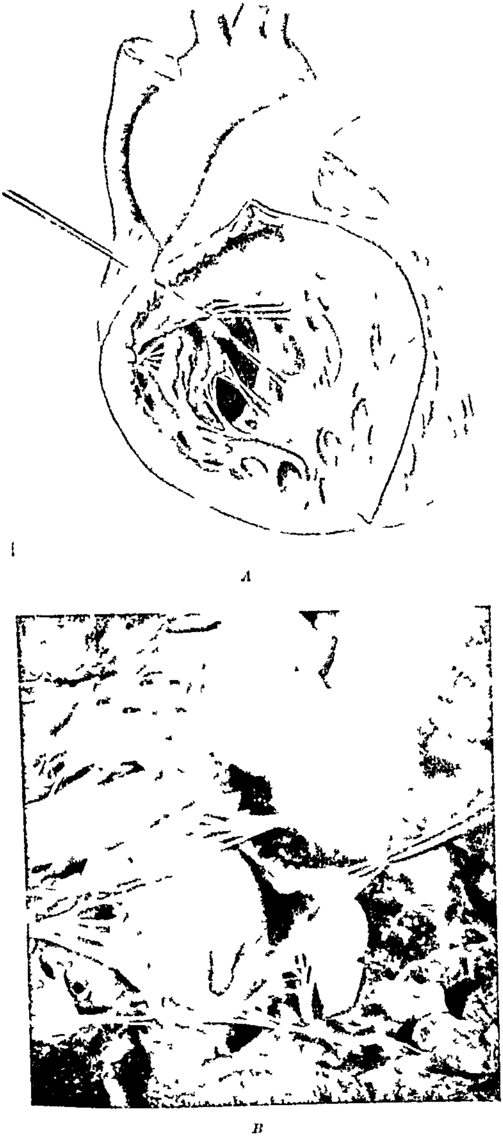


Fig 3 —Defect of the posterior septum (Rokitansky's Group B) *A* is an artist's version of a defect in the posterior ventricular septum and *B* shows a photograph of a similar defect taken from a view similar to that seen through a surgical cardiotomy in the right ventricle In this view the pulmonary valve cusps are visible at the top of the photograph and the manner in which the septal and posterior leaflets of the tricuspid valve tend to hide partially the margins of the defect is quite evident

of the position or origin of the great vessels. These malpositions range all the way from partial to complete transposition of the great vessels. The septal defect is usually located in or near the membranous area but, of course, may occur elsewhere as well. Certainly, because of the malposition and rotation of the great vessels, considerable variation is induced in the site of the septal defect. Fig. 5 is a representative specimen of the group. This photograph shows complete transposition of the great vessels and a ventricular septal defect. The right atrioventricular valve is apparent in the left foreground and just above its apex appears the septal defect. In the upper center of the field may be seen the aorta as it exits from this anterior ventricle

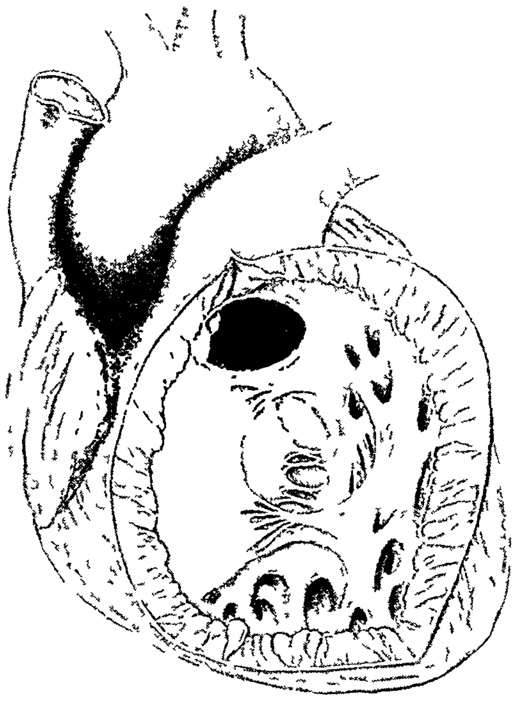


Fig 4 —Absence of the entire anterior septum (Rokitansky's Group C 1) This type of anterior septal defect with normal caliber of the arterial trunks is diagrammatically shown in this plate Note the proximity of the aortic and pulmonary leaflets

A discussion of this group is included primarily for completeness of the classification. Two patients with this type of ventricular septal defect have been operated upon as part of an attempt at complete corrective surgery for complete transposition of the great vessels[17]

Group C, 2a, beta: *Defect in the posterior part of the anterior septum with anomalous position of the arterial trunks and stenosis or atresia of the pulmonary artery (tetralogy of Fallot):* Under this heading, Rokitansky has grouped those defects associated with an enlarged, dextraposed aorta and stenosis or atresia of the pulmonary artery. Thirteen years later, Fallot[18] also described the pathologic anatomy of these defects and correlated it with the clinical findings in patients possessing that combination of congenital intracardiac anomalies. Since his classic description in 1888 this tetralogical association has attained common usage whenever reference is made to that pathologic complex.

The significant abnormalities in this syndrome are the ventricular septal defect and stenosis or atresia of the pulmonary artery. The positional alterations in the aorta and pulmonary artery come in part from a faulty partitioning of the truncus arteriosus by the septum aorticum,[12, 13] and in part are acquired secondarily, after birth, from the disproportionate blood flow through these respective vessels. The result is an inequality in the size of these two vessels which, together with certain other factors,[7] contributes to the impression that the aorta is overriding. Thereafter, the aorta encroaches upon this area normally occupied by the posterior portion of the anterior septum (see Fig. 1, *D, 2-4*). Although the pulmonary artery is always the smaller of the two vessels its actual size may vary from an atretic cord or hypoplastic vessel to that of an artery scarcely diminutive to the normal.

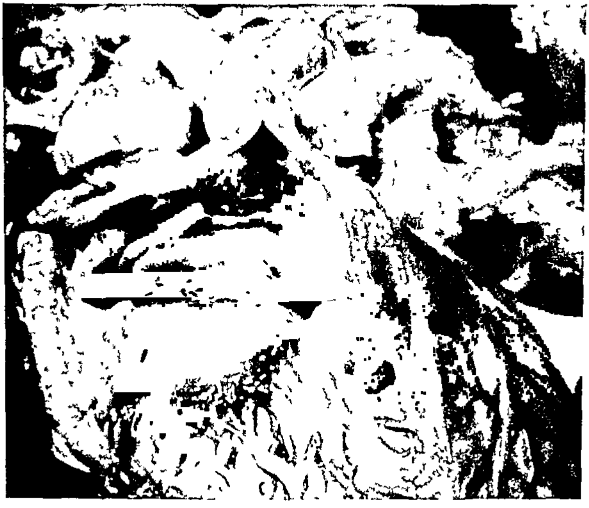


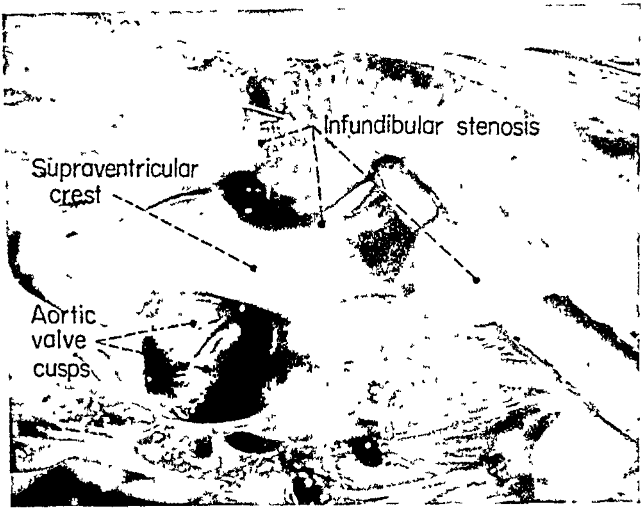
Fig. 5.—Defect in posterior part of anterior septum with anomalous position of the arterial trunks (Rokitansky's Group C, 2a, alpha). An example of the defects included in this group is the above photograph from a 4-month-old child dying of a complete transposition of the great vessels. The aorta, located anterior to the pulmonary artery, can be seen coming off the right (anterior) ventricle. The defect involving the posterior part of the anterior septum is seen in the center just above the commissure of the right atrioventricular (tricuspid) valve.

Associated with a slight malposition of the aorta, there is also an exaggerated prominence of the crista supraventricularis. The latter, quite like the pulmonary artery, may exhibit considerable variation in size. It can be completely absent, as described in Group C, 1b, or it may exist as a moderately thickened muscular ridge. In its extremely overdeveloped form, it appears as a markedly hypertrophied muscular mass obstructing the right ventricular outflow tract to a pinhole. In the latter instance, it usually forms the floor of a rather well-developed infundibular chamber proximal to the orifice of the pulmonary artery. At times the encroachment through hypertrophy is capable of completely obliterating this region. Finally, through an asymmetrical division of the truncus arteriosus, there is frequently a deficiency in the number of valve cusps, resulting not uncommonly in either a bicuspid aortic or pulmonic valve.

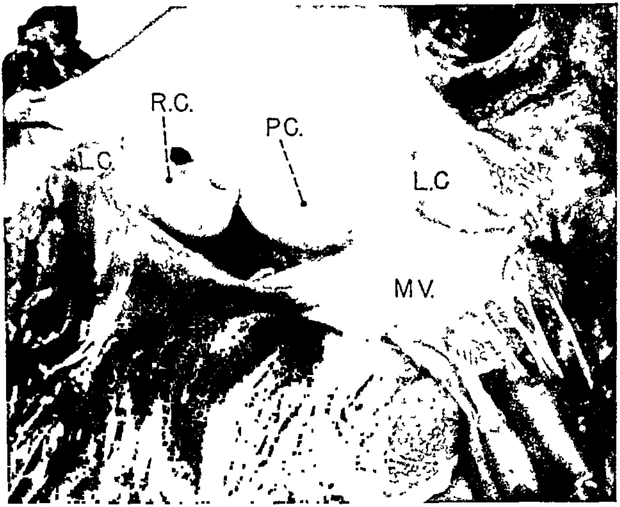
The septal defects noted in this group are generally located in the membranous area of the septum and have been generally large in size. However, despite the similarity in location they differ considerably in appearance from the pure membranous type of defect. Characteristically, they show an extremely narrow upper margin immediately adjacent to the origin of the aortic cusps from the fibrous base of the aortic ring (Fig. 6, *B*). The remaining margin is predominently muscular as a rule but occasionally may consist too of a rather narrow shelf of fibrous tissue. This close proximity of the aortic cusps is seen in Fig. 6, *A*, an autopsy specimen with the view from the right ventricular side. In the lower left-hand corner lie the tricuspid valve orifice and leaflets while just above and medial to this is the septal defect through which appear two of the aortic cusps. This patient died before corrective surgery could be performed and therefore the thickened crista supraventricularis is shown as an intact ridge which traverses the right ventricular outflow tract. In this field on the right of the crista is a broad band of muscle extending from the septum to the anterior ventricular wall. The tip of a hemostat passes beneath this structure. This configuration has been found in the majority of cases in this group. Unquestionably, it serves to obstruct blood flow into the pulmonary artery. We have excised it therefore since its removal augments substantially the volume of blood moving through the outflow tract. Fig. 6, *B* depicts the septal defect as seen from the left ventricular aspect. Again, the proximity of the right and posterior aortic valve cusps to the typical crescent shape of the defect is apparent.

To date 33 patients with tetralogy of Fallot have been operated upon. In 29, the location of the septal defect was of this type (C, 2a, beta). Three of the other cases had deficiencies of the entire anterior septum as previously noted; the fourth patient, who preoperatively was thought to have a tetralogy of Fallot, had an atrioventricularis communis defect of the septum with deformed mitral leaflets and a severe pulmonary valvar stenosis. One patient with tetralogy of Fallot in this series had complete atresia of the origin of the main pulmonary artery (see Fig. 11, *A*). She continues to do well one and one-half years postoperatively after correction of that anomaly also.[5] In this group, just one patient with tetralogy of Fallot had only valvular stenosis. Twenty-seven had infundibular stenosis and 5 had both types of obstruction to pulmonary outflow. Three patients had other congenital cardiovascular abnormalities; one had an associated patent ductus arteriosus which was divided, one had a left superior vena cava entering the coronary sinus, and one also had a primum defect of the interatrial septum. Right aortic arch was present in approximately one-third of these patients.

It is important to emphasize, for surgeons and cardiologists alike, the fact that the position of the aorta has not presented any important obstacle to curative surgery for Fallot's tetralogy, even in its severest forms encountered in infancy.

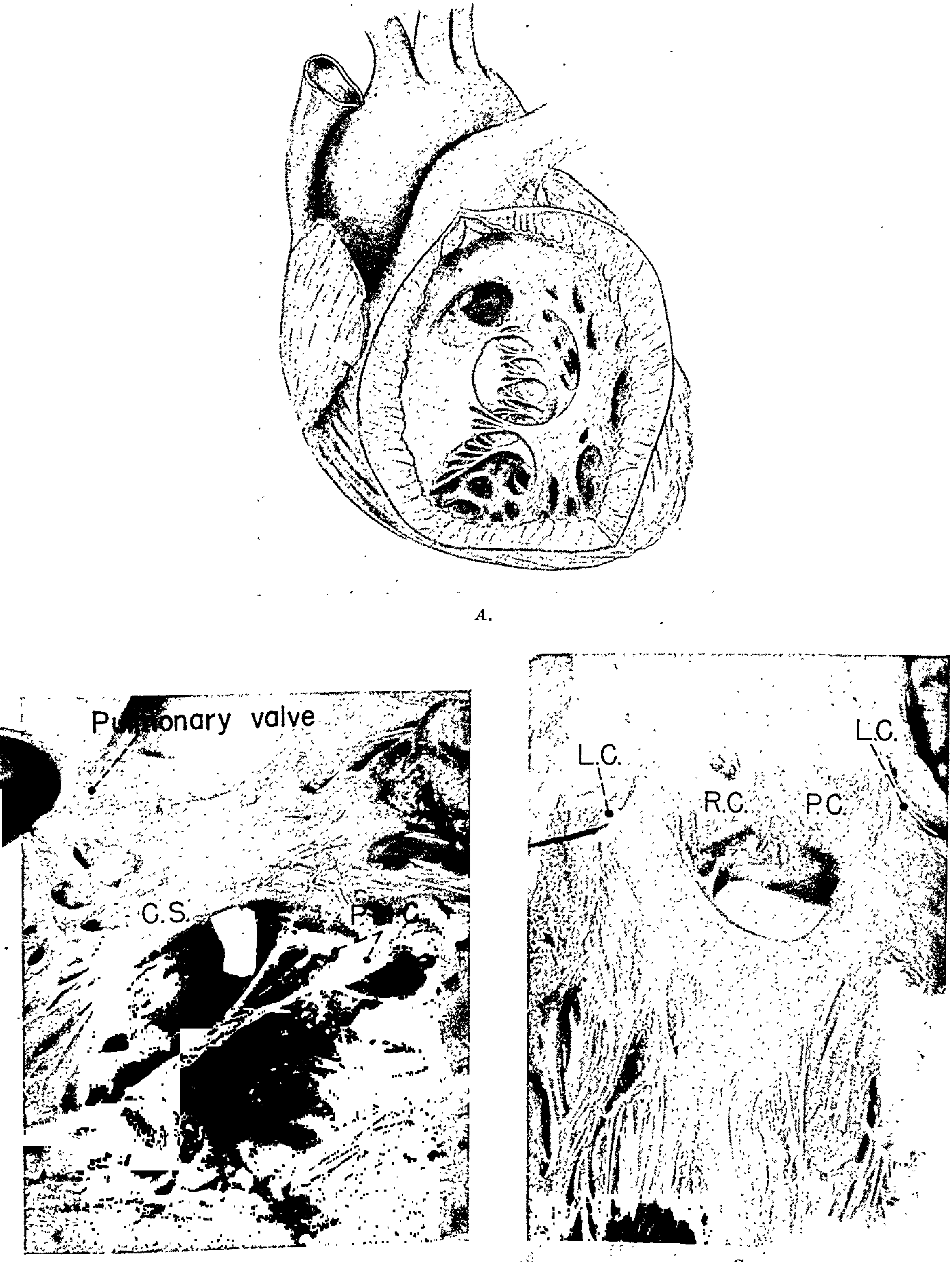


A.



B

Fig 6—Defect in posterior part of anterior septum with anomalous position of the
Group C, 2a, beta) This is an example of a tetralogy of Fallot
is a close-up view from within the right ventricle looking toward
flow tracts In the upper portion is seen the markedly stenotic
1 to the right a large muscular trabeculae (tented up by the
s been found obstructing the outflow tract in this region and
ie of corrective surgery. In the lower central portion are seen
high interventricular defect and in B the wide crescent-shaped
ventricular side and demonstrates the close proximity of
efect. The aorta is seen arising entirely from the left.
The right aortic cusp (R C) and the posterior cusp (P C) of the aortic valve are



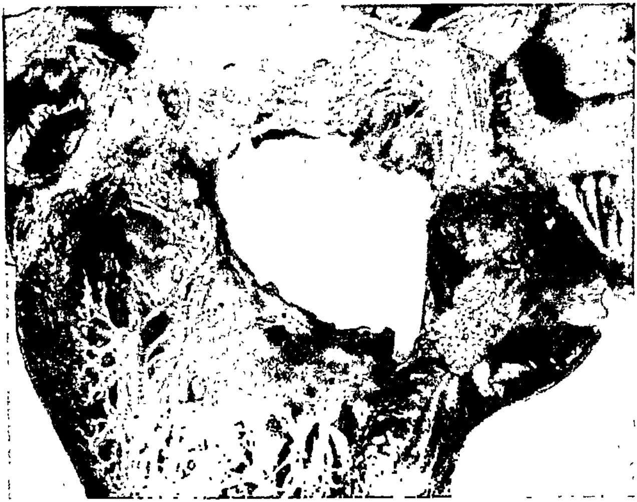
A.

B.

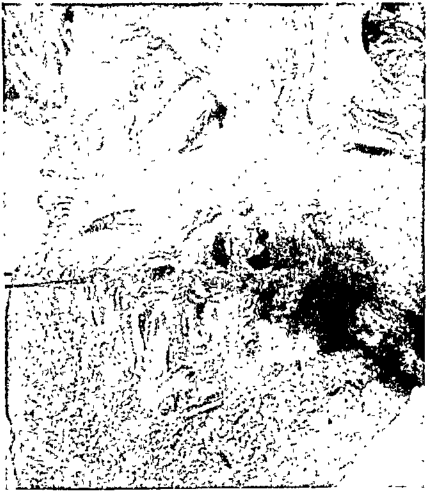
C.

Fig. 7.—Defects in the membranous septum
(Rokitansky's Group C, 2b). *A* demonstrates dia
Note how the defect margin extends beneath and
*B* is a right ventricular photograph of such a
to the crista supra-ventricularis (C.S.) and th
*C* is a view of the same defect viewed from
defect to the right aortic cusp (R.C.) and the p
valve is evident.

of
m.



*D.*



*E.*

Fig. 7 (Cont'd).—*D* indicates a defect of this type viewed from the left side, occurring in a 4-year-old boy. Defects in this area grow disproportionately larger with time. *E* shows a somewhat unusual type of membranous septal defect found in a 5-week-old infant. This is viewed from the right ventricular side and in the central portion shows an aneurysmal dilatation of the membranous septum and a perforation through the septal leaflet of the tricuspid valve, thus creating a direct communication between the left ventricle and the right auricle. (See text for details.)

Group C, 2b: *Defect in the posterior part of the anterior septum with normal position of the arterial trunks (defects of the membranous septum):* A defect in the membranous portion of the ventricular septum with normal location of the aorta and pulmonary artery was the most frequent congenital intracardiac malformation encountered in this series. These 71 abnormal ostia varied in diameter from 5 mm. to 6 cm. (Fig. 7, *D*) depending upon the amount of adjacent muscular septum that also was deficient.

As sketched in Fig. 7, *A,* the posterior margin can be difficult to expose for it lies behind the papillary muscle of the conus, concealed by the anterior commissure of the tricuspid valve. In reality no true defect margin need exist in this area. The edge of the septal hiatus merges with the annulus fibrosus of the tricuspid and mitral valves and this in turn diffuses into the aortic septum. Surgical exposure is also limited by attachments of the chordae tendineae to the inferior margin of the septal defect along its mid to anterior position. Occasionally, these chordae pass through the defect and insert on the left ventricular side of the septum. The usual characteristics of this lesion are shown in Figs. 7, *B, C, D.* Plates *B* and *C* are photographs of a specimen* viewed from the right and left ventricles, respectively. The septal hole is centrally located under the normally thin, broad, shelflike projection of the crista supraventricularis which here has an entirely different configuration from its appearance in a tetralogy of Fallot and hence it does not infringe on the pulmonary outflow tract. Fig. 7, *C,* a photograph of the same specimen, seen from the left ventricle, demonstrates [illegible]e relationship of the aortic valve to this membranous septal defect. Char[illegible]cteristically, in this type of defect, the aortic cusps are located a few millimeters farther away from the upper edge of the hole than when this kind of anomaly exists in a tetralogy. In the latter, this edge is usually very close to the aortic cusps. These relationships are of considerable importance in the placement of sutures during the repair.

Of the seventy-one membranous septal defects seen in this series of patients, four have had a definitely different appearance. The ventricular defect in each instance was in the usual location but arose from an aneurysmal dilatation of, and perforation in, the membranous septum. One infant had in addition a perforation through the origin of the septal leaflet of the tricuspid valve. This resulted in a direct shunt from the left ventricle into the right auricle. A defect similar to this has been described by Rokitansky,[12] and Edwards and associates.[19] The specimen from this case is s[illegible] [illegible]ig. 7, *E.* T[illegible] [illegible] was opened so as to expose the right auricular a[illegible] [illegible] of center lies the aneurysmal pouch and [illegible] through which can be seen the margins of [illegible]ta background. This unusual lesion produce[illegible] This infant though merely 6 weeks old was [illegible] a last resort.

---

*This patient, J. D., age 16 weeks, died in the [illegible]

Twenty-six patients in this group had other cardiovascular anomalies. Among these there were five instances of left superior venae cavae. Four patients had a patent ductus arteriosus which was transected at the time the other corrective surgery was performed. Three had associated atrial septal defects which were likewise repaired. Mitral stenosis was found in one patient with a defect in the membranous ventricular septum. He underwent commissurotomy under direct vision at the time the ventricular septal defect was closed. The remaining 2 patients had coarctation of the aorta.

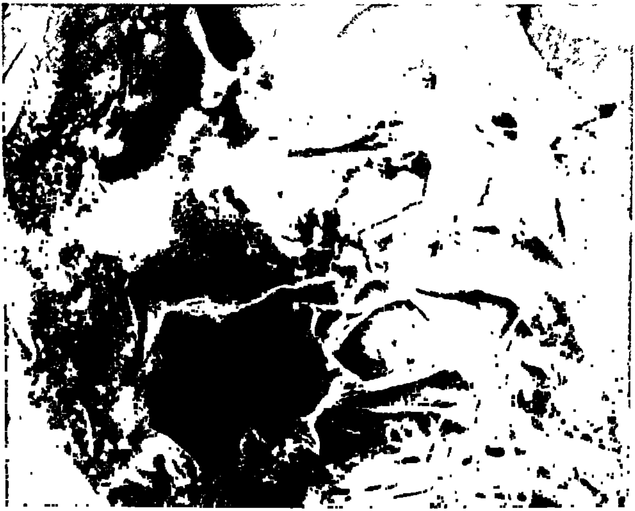
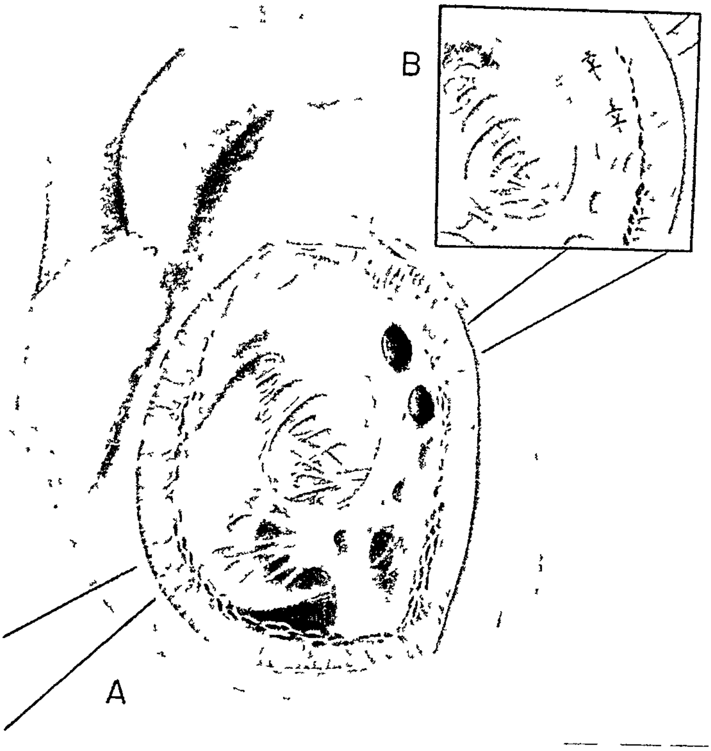


Fig. 8.—Defect in the anterior part of the anterior septum (Rokitansky's Group C, 3). Note the proximity to the pulmonary valves

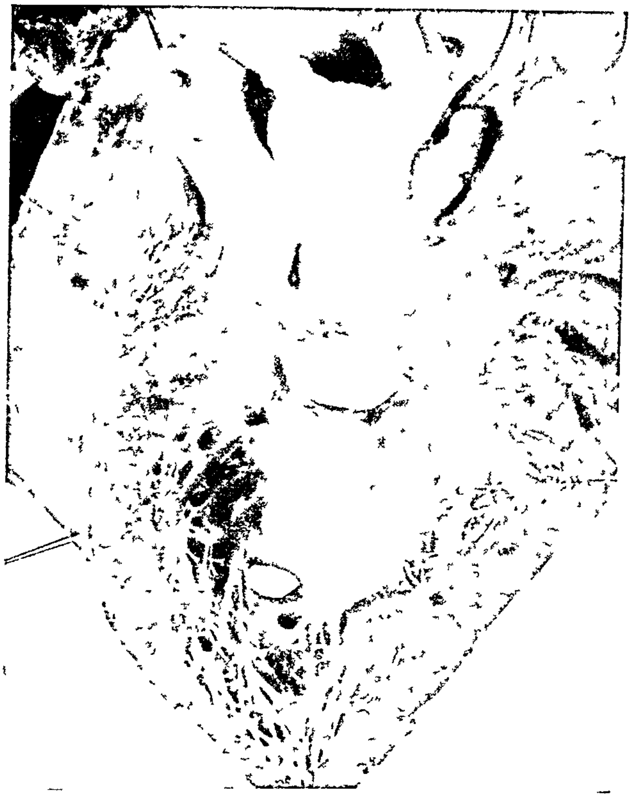
Group C, 3: *Defect in the anterior part of the anterior septum:* Defects of the muscular portion of the anterior ventricular septum occur in the area between *1* and *2* in Fig. 1, *C*. This type defect is usually located close to the anterior ventricular wall just below the pulmonary valves (Fig. 8). Those we have found were 4 to 15 mm. in diameter. Uniformly, they have had a muscular margin inferiorly. In this defect, similar to that mentioned above in regard to absence of the entire anterior septum, the aortic valve (left anterior cusp) and the pulmonary valve leaflets are separated by only a narrow (2 to 4 mm.) margin. Hemodynamically also, they unite the outflow tracts of the right and left ventricles (similar to Group C, 1) and hence provoke severe systemic effects. The four met with in this series were readily visualized and the patients tolerated the surgical closure well.

Group D: *Defect in Unusual Positions (Including Muscular Defects).*—

Three patients have been found at operation to have this type of deficiency of the muscular portion of the ventricular septum. Two had associated cardiac



*A.*



*B.*

Fig 9.—Defects in unusual locations (including muscular defects) (Rokitansky's Group D). *A,* multiple muscular defects occurring as the sole defects in a seriously ill 5-month-old infant. Successfully repaired in 1954, utilizing controlled cross circulation. *B,* specimen from a 6-year-old boy which shows a moderate-sized muscular defect low in the septum in addition to a large membranous septal defect as seen from the left ventricular side.

lesions. One had a large, yet correctable and typically located, defect in the membranous area as well. The other patient had mitral stenosis too, which was relieved under direct vision after closure of the ventricular defect. In all, the defects had friable muscular margins and their diameters ranged from 5 to 15 mm. The third patient had multiple muscular defects (Fig. 9, *A*). Frequently several trabeculae carneae overlay the right ventricular orifice of the defect thus creating an initial impression that a cluster of several small defects exists. However, once these trabeculae are incised it is apparent that *these ostia coalesce into a single opening perforating the septum.* Occasionally, as we have noted, the holes may truly be multiple (Fig. 9, *A*). The more common sites are along the muscular septum at almost any level subjacent to the anterior ventricular wall but on occasion we have found them along the posterior ventricular wall. When present in the central portion of the muscular septum they have been near the apex. The specimen seen in Fig. 9, *B* demonstrates this situation as seen from the left ventricle. Here, the low muscular septal defect is associated with a rather large membranous opening as well. That particular combination occurred once in the operative series and both defects were successfully closed.

Group E. *Defect in Anomalous Septa.*—

Rokitansky has included under this heading a multiplicity of defects involving malposed septa, for example, those associated with a rudimentary ventricle and various gradations of transposition as well as the situation in which both great vessels come from either the large or small ventricle in combination with varying degrees of anomalous venous inflow to the heart. We have recognized only one patient in this category among our operative series, but have seen other examples at autopsies. Our patient had a "corrected" transposition by virtue of the anomalous septum together with large ventricular and atrial (secundum) defects.

Group F. *Acquired Ventricular Septal Defects.*—

Acquired ventricular septal defects can develop after a crushing chest injury, bacterial endocarditis or from coronary infarctions. We have had an operative experience with the former.[9] The patient sustained multiple rupture sites in the ventricular septum following compression of the thorax under a rolling truck wheel. The three defects were located in the muscular septum near the apex of the heart. They possessed heavily scarred margins, were easily approximated by silk stitches and the patient remains well one and one half years, postoperative. This group is an addition to Rokitansky's classification that seemed indicated, for completeness, in light of clinical experience.

## ASSOCIATED DEFECTS

Among this group of patients who underwent direct surgical repair of their ventricular septal defects, a number were found to have other associated cardiovascular malformations. These have been recorded in Table III. As a result of this experiment we have come to believe that it is probably wise to correct the coexisting anomaly either primarily or coincidentally with repair of the ventricular defect. This, of course, is not applicable to endocardial fibroelastosis,

Table III. Associated Cardiac Malformations Encountered During Reparative Surgery Ventricular Septal Defects Including Tetralogy of Fallot

| PRIMARY DEFECT | NUMBER PATIENTS | MULTIPLE VSD | ATRIAL SEPTAL DEFECT | PATENT DUCTUS ARTERIOSUS | COARCTATION | ENDOCARDIAL FIBROELASTOSIS | CORRECTED TRANSPOSITION | LEFT SUPERIOR VENA CAVA | DEXTRA-CARDIAC |
|---|---|---|---|---|---|---|---|---|---|
| Ventricular septal defect | 87 | 3 | 3 | 4 | 2 | 7 | 1 | 5 | 1 |
| Tetralogy of Fallot | 33 | 0 | 1* | 1 | 0 | 1 | 0 | 1 | 0 |
| Total | 120 | 3 | 4 | 5 | 2 | 8 | 1 | 6 | 1 |

*Ostinum primum.

corrected transposition, dextrocardia, or left superior vena cava However, it is especially important for the surgeon to recognize the presence of this latter abnormality for unless it is occluded during the by pass period the operative site is often unnecessarily obscured by this venous return Also, particularly if this accessory superior vena cava is of any significant size, it is better not to occlude it but rather to cannulate it in order to minimize possibility of cerebral congestion if cross connections are lacking

Similarly, aside from the physiologic undesirability of leaving an unrecognized coarctation of the aorta or patent ductus arteriosus uncorrected, overlooking the latter lesion at the time of corrective intracardiac surgery may result in catastrophic hemorrhage when the heart is opened

### SURGICAL MANAGEMENT

Spatial limitations require that a detailed discussion of this important consideration be deferred Currently we find three methods useful These are (1) direct suture, (2) Ivalon pledget, and (3) circumferential patch These types of closure are portrayed diagrammatically in Fig 10 The particular anatomic configuration found, when the heart is open, decides the choice The frequency with which each type was used has been summarized in Table IV The distribution of methods used is somewhat askew, however, because initially many defects were closed by direct suture whereas now we would probably favor the use of an Ivalon pledget or a circumferential patch for many of these cases

TABLE IV METHOD OF REPAIR OF VENTRICULAR DEFECTS

| TYPE OF DEFECT | NUMBER PATIENTS | STITCHES ONLY | PLEDGET | PATCH |
|---|---|---|---|---|
| B | 5 | 2 | 3 | 0 |
| C 1 a (without pulmonary stenosis) | 1 | 1 | | |
| b (with pulmonary stenosis) | 3 | 1 | 0 | 2 |
| C 2 a (Alpha) | 2 | 1 | 1 | 0 |
| (Beta) | 30 | 10 | 17 | 3 |
| C 2 b | 71 | 37 | 32 | 2 |
| C 3 | 4 | 1 | 3 | 0 |
| D | 3* | 2 | 1 | 0 |
| E | 1 | 0 | 1 | 0 |
| F Traumatic VSD | 1 | 1 | 0 | |
| Total | 121 | 56 | 58 | — |

*One of these also had a C 2b type defect

*Right Ventricular Outflow Tract Roof for Patients With Tetralogy of Fallot* —

The curative procedure for tetralogy of Fallot has completely replaced the palliative shunt procedure in this clinic The achievement of this more desirable treatment for these patients involves two considerations, namely, closure of the ventricular septal defect and removal of the pulmonary stenosis The anatomy and techniques of closure of their ventricular septal defects have been discussed above

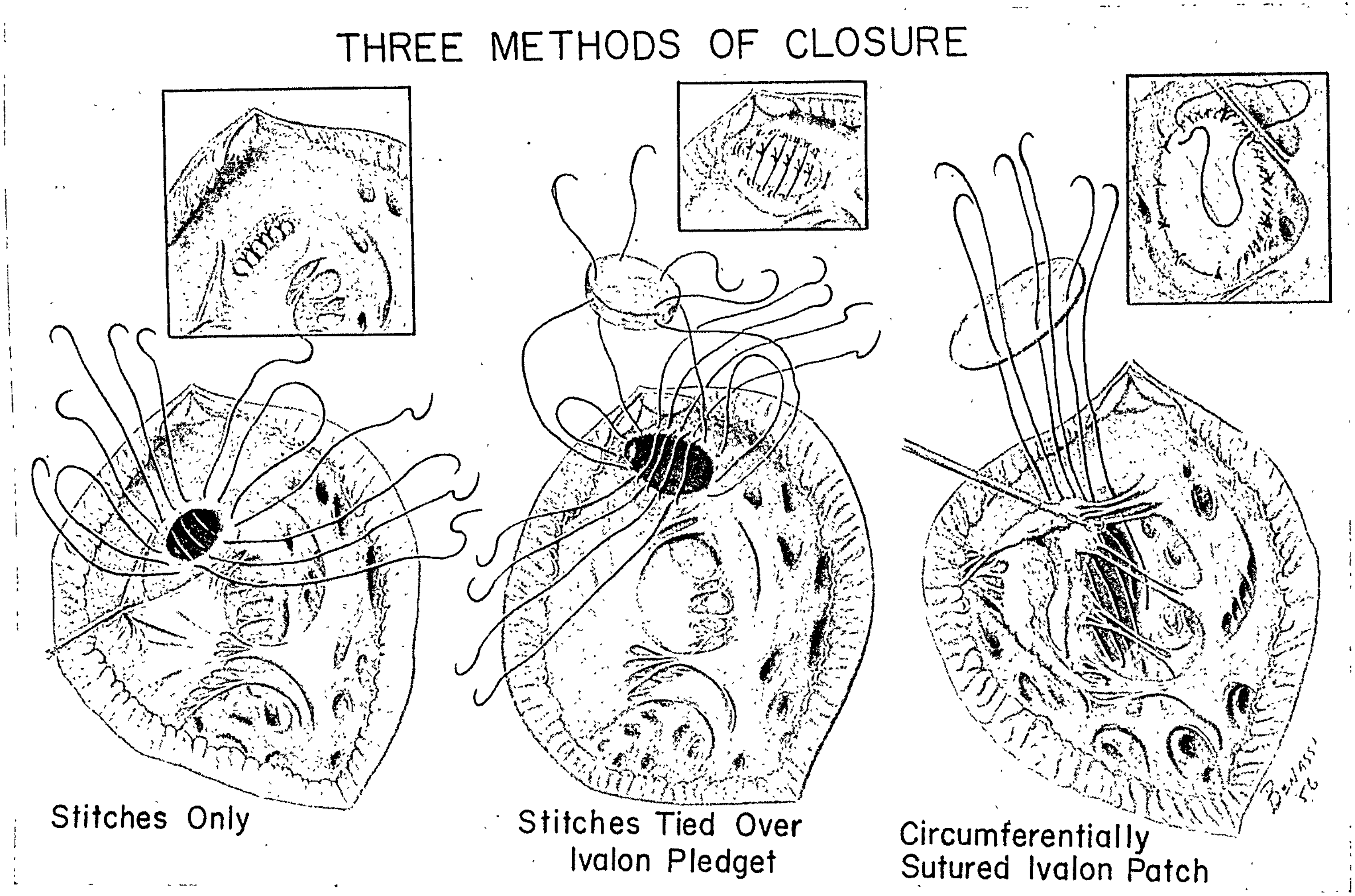

THREE METHODS OF CLOSURE
Stitches Only
Stitches Tied Over Ivalon Pledget
Circumferentially Sutured Ivalon Patch
Denassi 56

Fig. 10.—The methods of closure of ventricular septal defects. Silk (3-0) with a double-armed swedged-on needle was the suture material utilized in all instances.

Pulmonary valvulotomy and/or infundibulectomy (Fig 11, *A*, and *B*) has been satisfactory for many of these patients operated upon,[7, 20] but one of the important technical innovations has been the use of a teardrop-shaped piece of compressed Ivalon sutured into the upper portion of the right ventriculotomy

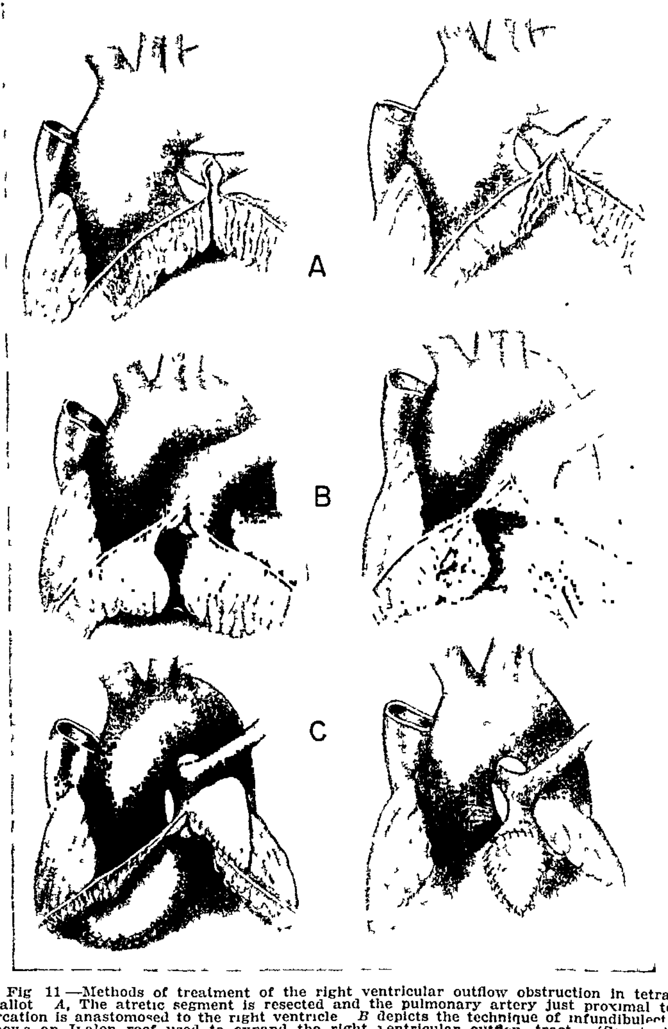


Fig 11 —Methods of treatment of the right ventricular outflow obstruction in tetralogy of Fallot *A*, The atretic segment is resected and the pulmonary artery just proximal to its bifurcation is anastomosed to the right ventricle *B* depicts the technique of infundibulect *C* shows an Ivalon roof used to expand the right ventricular outflow tract (See text details )

(Fig. 11, *C*) to enlarge further this outflow tract in the severe tetrads where it is not anatomically possible to excise a sufficient amount of muscle to accomplish this goal.

## DISCUSSION

Our identification of the particular types of ventricular septal pathology seen in this series of cases has been greatly facilitated both by the use of Rokitansky's classification and from familiarizing ourselves with these defects in terms of their embryologic development. His classification has required but few modifications in order to provide a comprehensive coverage, yet still remain a conveniently workable method.

We recognize that the incidence shown in Table II for these various types of defects is unlikely to be a precise reflection of their natural occurrence rate. As indicated previously, the patients in this series were selected on the basis of their deteriorating clinical course. All persons operated upon were, or at some time had been, severely ill and none was asymptomatic. Except for those in tetralogy of Fallot groups, all had pulmonary hypertension. In the majority of them the pulmonary artery systolic pressure was greater than 70 per cent of the aortic systolic pressure. Many suffered repeatedly from severe respiratory infections and those few who were chosen, *because* of their peripheral arterial desaturation, emphasize their desperate need for corrective surgery. The compelling operative indications among the tetralogy of Fallot patients have already been cited.

The site of the most common septal defect encountered in this series was in the pars membranacea. Too, the clear-cut preponderance in frequency of the membranous defects over the other forms of ventricular septal defects substantiates our belief that these lesions are invariably detrimental to the patient. Undoubtedly, from their location a strong pulsatile thrust is imparted to the pulmonary artery and as a consequence severe proliferative changes frequently develop in the arterioles of this vascular bed at an early age.

While there has been some correlation between the size of the defect and the degree of clinical disability in patients with isolated ventricular defects, a factor, at least as important in this regard, has been the precise anatomical relationship of the defect to the orifice of the pulmonary artery. Thus, even relatively small defects (whether they be in the pars membranacea, in the anterior, or in the posterior ventricular septum), situated so that the pulsatile thrust of the bolus of the blood from the left ventricle projects directly into the pulmonary artery, are hemodynamically more deleterious than considerably larger defects situated so that the left ventricular jet strikes the opposite ventricular (right) wall in such a way as to create turbulence in the outflow tract of this ventricle. The consequent increased resistance due to this turbulence creates, in effect, a functional pulmonic stenosis tending to dampen the physiologic consequences of the defect.

In view of the fact that corrective surgery is now possible and since it is an historical fact that these patients with ventricular septal defects rather uniformly are seriously ill and have a severely contracted life expectancy, it would therefore seem pertinent currently to balance for each patient the increasing hazard of procrastination against the decreasing operative risk.

We wish to express our appreciation to Dr. Bernard Hoelscher of the Department of Surgery, University of Minnesota, for his aid in the translation of the original papers of Rokitansky and Spitzer.

## REFERENCES

1. Warden, H. E., Cohen, M., Read, R. C., and Lillehei, C. W.: Controlled Cross Circulation for Open Intracardiac Surgery. Physiologic Studies and Results of Creation and Closure of Ventricular Septal Defects, J. Thoracic Surg. 28: 331-343, 1954.
2. Lillehei, C. W.: Controlled Cross Circulation for Direct Vision Intracardiac Surgery. Correction of Ventricular Septal Defects, Atrioventricularis Communis, and Tetralogy of Fallot, Postgraduate Med. 17: 388-396, 1955.
3. Lillehei, C. W., Cohen, M., Warden, H. E., Ziegler, N., and Varco, R. L.: The Results of Direct Vision Closure of Ventricular Septal Defects in 8 Patients by Means of Controlled Cross Circulation, Surg., Gynec. & Obst. 101: 446-466, 1955.
4. Lillehei, C. W., Cohen, M., Warden, H. E., and Varco, R. L.: The Direct Vision Intracardiac Correction of Congenital Anomalies by Controlled Cross Circulation. Results in 32 Patients With Ventricular Septal Defects, Surgery 38: 11-29, 1955.
5. Lillehei, C. W., Cohen, M., Warden, H. E., Read, R. C., Aust, J. B., DeWall, R. A., and Varco, R. L.: Direct Vision Intracardiac Surgical Correction of the Tetralogy of Fallot, Pentalogy of Fallot, and Pulmonary Atresia Defects. Report of First Ten Cases, Ann. Surg. 142: 418-455, 1955.
6. Lillehei, C. W.: Discussion of Kay, Zimmerman, and Cross: Results of Direct Vision Intracardiac Surgery Utilizing Controlled Cross Circulation, J. Thoracic Surg. 30: 452, 1955.
7. Lillehei, C. W., Cohen, M., Warden, H. E., Read, R. C., DeWall, R. A., Aust, J. B., and Varco, R. L.: Direct Vision Intracardiac Surgery by Means of Controlled Cross Circulation or Continuous Arterial Reservoir Perfusion for Correction of Ventricular Septal Defects, Atrioventricularis Communis, Isolated Infundibular Pulmonic Stenosis, and Tetralogy of Fallot, Proceedings of the International Symposium of Cardiovascular Surgery, Henry Ford Hospital, Philadelphia, 1955, W. B. Saunders Company, pp. 371-392.
8. Warden, H. E., Read, R. C., DeWall, R. A., Aust, J. B., Cohen, M., Ziegler, N., Varco, R. L., and Lillehei, C. W.: Direct Vision Intracardiac Surgery by Means of a Reservoir of "Arterialized Venous" Blood: Description of a Simple Method and Report of the First Clinical Case, J. Thoracic Surg. 30: 649-656, 1955.
9. Campbell, G. S., Crisp, N. W., and Brown, E. B., Jr.: Total Cardiac By-Pass in Humans Utilizing a Pump and Heterologous Lung Oxygenator (Dog Lung), Surgery 40: 364-371, 1956.
10. Lillehei, C. W., DeWall, R. A., Read, R. C., Warden, H. E., and Varco, R. L.: Direct Vision Intracardiac Surgery in Man Using a Simple Disposable Artificial Oxygenator, Dis. Chest 24: 1-8, 1956.
11. DeWall, R. A., Warden, H. E., Read, R. C., Gott, V. L., Ziegler, N., Varco, R. L., and Lillehei, C. W.: Surg. Clin. North America, Philadelphia, 1955, W. B. Saunders Company, pp. 1025-1034.
12. Rokitansky, K. F.: Die Defecte der Scheidewande des Herzens, Wein, 1875, Wilhelm Braumuller.
13. Spitzer, A.: Über den Bauplan des Normalen und Missbildeten Herzens, Virchow Arch. 243: 81-271, 1923.
14. Hamilton, W. J., Boyde, J. D., and Mausman, H. W.: Human Embryology, Baltimore, 1952, Williams & Wilkins Company.
15. Patten, B. M.: Human Embryology, Philadelphia and Toronto, 1953, Blakiston Company.
16. Abbott, M. E.: Atlas of Congenital Cardiac Disease, American Heart Association, 1936.
17. Gott, V. L., Gonzalez, J. L., Zuhdi, M. N., Varco, R. L., and Lillehei, C. W.: Retrograde Perfusion of the Coronary Sinus for Direct Vision Aortic Surgery. Experimental Studies and Application to Reparative Surgery for the Aortic Valve, Ruptured Sinus of Valsalva, Aortic-Pulmonary Septal Defect, and Complete Transposition of the Great Vessels in Man, Surg., Gynec. & Obst. (To be published, February, 1957.)
18. Fallot, A.: Contribution A l'Anatomie pathologique de la maladie bleue, Marseille Med. 25: 77, 1888.

19. Edwards, J. E., Dry, T. J., Parker, R. L., Bertro, H. B., Wood, E. H., and Bulbulian, A. H.: An Atlas of Congenital Anomalies of the Heart and Great Vessels, Springfield, Ill., 1954, Charles C Thomas, publisher.
20. Lillehei, C. W., Cohen, M., Warden, H. E., and Varco, R. L.: Complete Anatomical Correction of the Tetralogy of Fallot Defects. Report of a Successful Surgical Case, Arch. Surg. 73: 526-531, 1956.

## DISCUSSION

DR. DENTON A. COOLEY, Houston, Texas.—I rise to congratulate Drs. Warden, Lillehei, and their associates on this outstanding contribution. The pathologic anatomy of ventricular septal defects must vary more widely than had been previously appreciated. Recently we encountered at operation a defect of the membranous portion of the ventricular septum which resulted in a communication between the left ventricle and the right atrium without entering the right ventricle at all.

Another patient upon whom we recently operated was 49 years old and about ten weeks previously had suffered an extensive posteroseptal myocardial infarction. About one week later, spontaneous perforation of the ventricular septum occurred. At open cardiotomy, using the cardiac by-pass, a defect about 3 cm. in diameter was discovered in the posterior muscular portion of the septum, partly hidden by the septal leaflet of the tricuspid valve. In order to expose the defect, two papillary muscles of the septal leaflet were divided and the defect was closed with interrupted silk sutures incorporating a polyvinyl sponge. This operation was done six weeks ago and the patient is still alive.

One may question the efficacy of such a procedure on the basis that healing would be retarded by the surrounding infarcted muscle. Actually the collateral blood supply to the ventricular septum is good and healing should be satisfactory. The rim of tissue around the defect in this case was quite firm, and I think that a direct suture without the sponge would have been satisfactory. I believe that, in view of the grave prognosis of acquired septal defect, surgical repair of the lesion should always be attempted if the patient's general condition will permit operation.

DR. HERBERT E. WARDEN (Closing).—I should like to thank Dr. Cooley for his remarks. I was afraid of the time limit, consequently I omitted from the presentation two slides which I believe demonstrate the type of anomaly he alluded to. This type has been described in Rokitansky's[12] works on septal defects in 1875 and also by Edwards and associates.[19]

(slide) (See Fig. 7, *E* in text.) This is a photograph of a heart, viewed from the right ventricular side, which shows such a defect. Here, in the center of the photograph, you will note the aneurysmal dilatation of the membranous septum has two perforations adjacent to its apex. In addition, you will note that these two perforations communicate with similar sized perforations in the septal leaflet of the tricuspid valve. The location of these defects, together with the physiologic data obtained at cardiac catheterization, indicated that during life there was a considerable shunt directly from the left ventricle into the right auricle.

(slide) This second slide is a photograph of the same heart viewed from the left ventricular side. Here will be noted the rather typical location of the membranous defect and through it can be seen the concavity of the aneurysmal dilatation of the membranous septum with the openings at its apex.

This is an autopsy specimen from a 4-week-old infant who died in the early postoperative period. This patient was in extremely poor clinical condition preoperatively which can be attributed to this very unusual type of defect. Thus far in this series of patients this is the only one of this type of defect we have encountered. However, 3 other patients with intact tricuspid leaflets, but with a similar type ventricular defect, have been seen.

# SURGICAL CORRECTION OF VENTRICULAR SEPTAL DEFECT: ANATOMIC AND TECHNICAL CONSIDERATIONS

JOHN W. KIRKLIN, M.D.,* HARRY G. HARSHBARGER, M.D.** (BY INVITATION),
DAVID E. DONALD, B.V.S., M.R.C.V.S.*** (BY INVITATION), AND
JESSE E. EDWARDS, M.D.**** (BY INVITATION)
ROCHESTER, MINN.

ANALYSIS and solution of the technical problems inherent in surgical closure of ventricular septal defects must be of concern to all engaged in corrective intracardiac surgery. Knowledge of the anatomy of the lesion and the methods of repair is necessary no matter what technique is used to permit the surgeon to work within the open heart. A study of these problems, initiated in the dissecting room and completed in the operating theater, yielded the results presently reported.

Extracorporeal circulation, utilizing a mechanical pump-oxygenator with a Gibbon-type stationary vertical screen oxygenator, was employed in the cases herein reported. A series of 81 patients operated on by this method has given evidence as to the soundness of the technique. The details of the pump-oxygenator,[1] the methods for cannulation and perfusion during operation,[2, 3] physiologic studies,[4] and mortality rates[4, 5] have been previously reported and will not be repeated herein.

## PATHOLOGIC ANATOMY

The concept of the ventricular septum and its defects developed in a previous report[6] from this institution is a logical beginning to an analysis of the challenge posed by repair of ventricular septal defects. The present route of surgical approach being through the right ventricle, the relations as seen from this aspect are particularly pertinent.

Ventricular septal defects may involve regions of the ventricular septum related to the ventricular outflow tracts or regions related to the inflow tracts. The outflow portion of the right ventricle, as defined in the study mentioned, lies between the pulmonary valve above and the nearest portion of the tricuspid valve below. The inflow portion lies posterior and caudal to the outflow tract. Major anatomic landmarks of the interior of the right ventricle are: (1) the tricuspid valve ring and leaflets, (2) the papillary muscle of the conus, (3) the crista supraventricularis, (4) the annulus fibrosus of the pulmonary valve, and (5) the apex of the ventricular septum (Fig. 1).

The papillary muscle of the conus is that point of the septal wall of the right ventricle into which insert the chordae tendineae from the left half of the

---

Read at the Thirty-sixth Annual Meeting of the American Association for Thoracic Surgery, Miami Beach, Fla., May 7 to 9, 1956.

The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

*Section of Surgery, Mayo Clinic and Mayo Foundation.

**Fellow in Surgery, Mayo Clinic and Mayo Foundation

***Research Associate, Mayo Clinic and Mayo Foundation.

****Section of Pathologic Anatomy, Mayo Clinic and Mayo Foundation.

anterior tricuspid leaflet and the anterior half of the septal leaflet of this valve. In some hearts, particularly those in which the right ventricular chamber is dilated, the chordae tendineae appear to insert directly into the septal wall rather than into a recognizable specialized papillary muscle. The crista supraventricularis presents on the floor of the right ventricular chamber, as seen surgically, between the papillary muscle of the conus below and posteriorly and the pulmonary valve above and anteriorly.

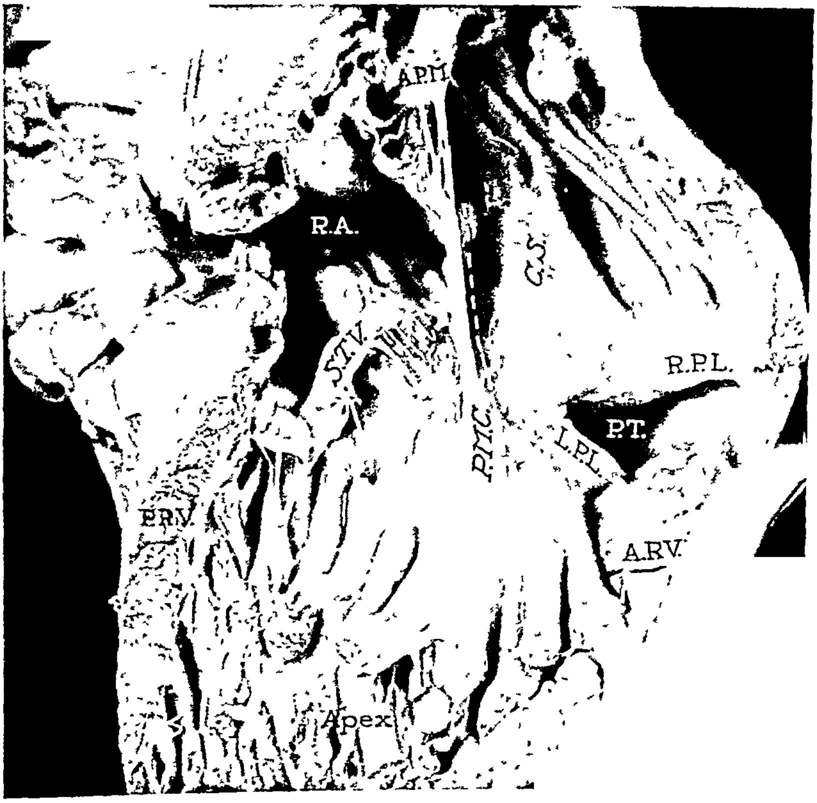


Fig. 1.—Normal heart with right ventricle opened. To view this specimen in the orientation seen at bilateral anterior thoracotomy, the photograph should be rotated 90 degrees counterclockwise. Key: *PT*, pulmonary artery; *LPL* and *RPL*, left and right pulmonary valve cusps; *CS*, crista supraventricularis; *PMC*, papillary muscle of conus ("conus muscle"); *APM*, anterior papillary muscle; *STV*, septal leaflet of tricuspid valve; *RA*, right atrium; *PRV* and *ARV*, posterior and anterior walls of right ventricle. The dotted circle represents the region of the membranous septum.

It is essential to recognize that most of the outflow tract of the right ventricle and the pulmonary valve lie more cephalad and anterior than do the outflow tract of the left ventricle and aortic valve when the heart is in situ. Thus, the crista supraventricularis overlies in part the root of the aorta.

Ventricular septal defects may be classified as indicated in Table I.

TABLE I. TYPES OF VENTRICULAR SEPTAL DEFECTS ENCOUNTERED IN 36 SURGICAL CASES

| TYPE OF DEFECT | CASES | |
|---|---|---|
| Defect related to ventricular outflow tracts | 27 | "High" |
| Inferior to crista supraventricularis | 25 | |
| Superior to crista supraventricularis | 2 | |
| Defect related to ventricular inflow tracts | 11 | |
| Beneath septal leaflet of tricuspid valve | 8 | |
| Near apex of muscular septum | 3* | "Low" |

*In one case the only defects (three) were in this area. In the other 2 cases, defects here were associated with "high" ventricular septal defects.

*Defects Related to Ventricular Outflow Tracts and Involving Region Inferior to Crista Supraventricularis.*—The region of the right ventricular outflow tract lying inferior to the crista supraventricularis is most commonly involved by ventricular septal defects. Defects in this location, when viewed from the left ventricular aspect, are seen to be immediately beneath the aortic valve cusps (Fig. 2).

These lesions are usually classified as "membranous ventricular septal defects," an inadequate descriptive name since they basically involve a large zone of muscular tissue adjacent to the membranous septum (Fig. 3). The size of the defect itself seems related mainly to the amount of this muscular portion of the ventricular septum that is absent. In some cases, the membranous portion of the ventricular septum is also lacking. In such instances, the septal leaflet of the tricuspid valve and the anterior leaflet of the mitral valve are continuous across the posterior border of the defect.

When the defects in this region are large and involve the septal tissue immediately below the annulus of the aortic valve, the aortic leaflets are seen to overhang the defect (Fig. 4). In all defects with this configuration, the annulus and leaflets of the aortic valve are very near the region where stitches for repair must be placed.

*Defects Related to Ventricular Outflow Tracts and Involving Region Superior to Crista Supraventricularis.*—The main features of such defects are their close relationship to the pulmonary valve and their lack of extension inferiorly and posteriorly beyond the crista supraventricularis. These defects lie immediately beneath not only the pulmonic valve but also the left aortic cusp (Fig. 5).

*Defects Involving Regions of Ventricular Septum Not Related to Ventricular Outflow Tracts.*—These defects are located entirely in the muscular wall of the ventricular septum and are related to the inflow or sinus portion of the ventricles. Two categories of such defects are recognized:

1. *Defects lying beneath septal leaflet of tricuspid valve:* These, like the previously discussed types, might be called "high" ventricular septal defects. While they lie in the upper or basal portion of the septum, they are not closely related to the aortic valve when viewed from the left ventricular aspect (Fig. 6). Here they lie near the posterior leaflet of the mitral valve.

2. *Defects in muscular septum near apex:* These are often termed "low" ventricular septal defects (Fig. 7). Fig. 8 shows diagrammatically the location of these types of defects.

*Conduction System.*—It has been observed at operation that trauma, either by a fixation forceps or a needle point, to the remnant of membranous septum or tissue lying at the postero-inferior margin of the ventricular defect may result in ventricular asystole or heart block for a prolonged period. This suggests that conduction tissue lies in this area.

The detailed relations of the conduction system to ventricular septal defects are yet to be investigated. Nevertheless, observations on conduction disturbances occurring during and subsequent to repair of ventricular septal defects support the view that the conduction system in hearts with such a

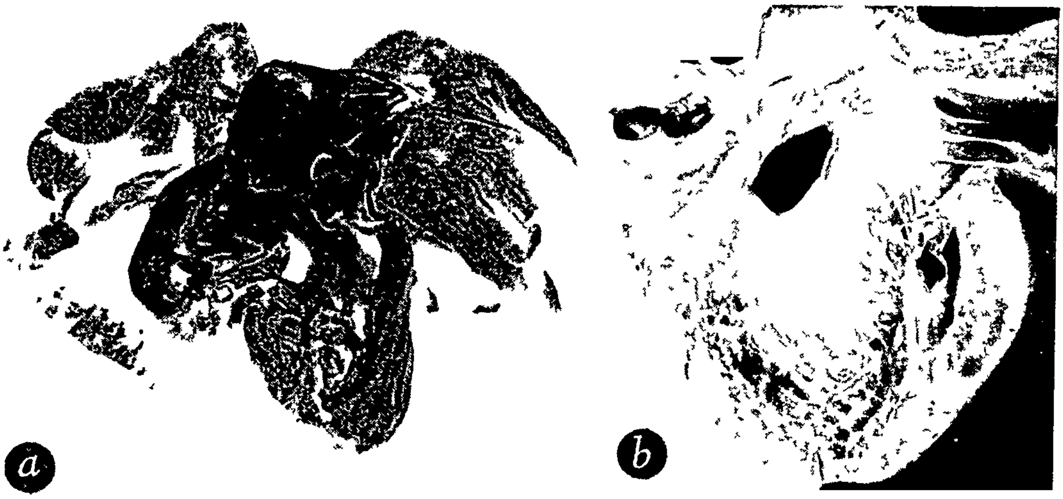


Fig 2 —*a*, Ventricular septal defect related to ventricular outflow tracts and lying posterior to crista supraventricularis, as viewed from the right ventricular aspect *b*, Same defect from left ventricular aspect. The upper margin of the defect, as viewed from this aspect, is formed by the annulus of the aortic valve

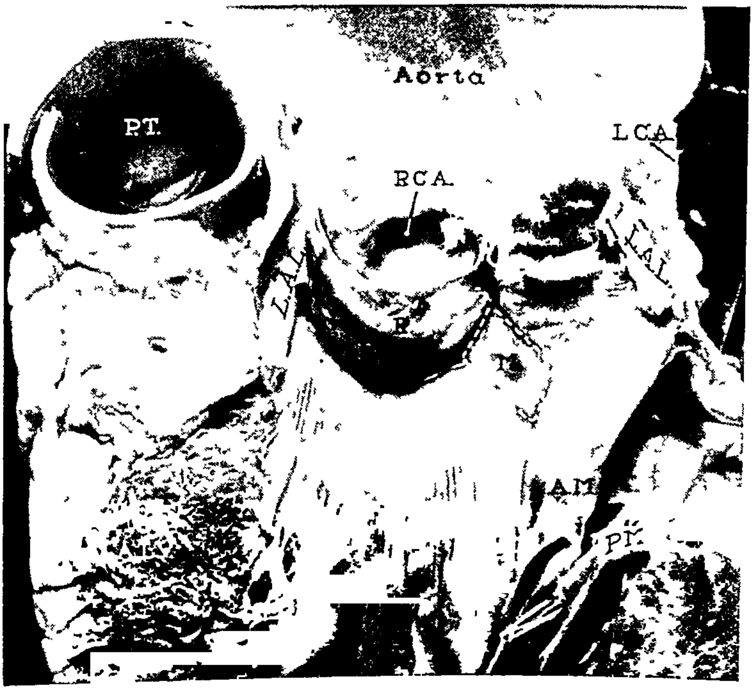


Fig 3.—Normal heart showing left ventricular aspect of septum. The dotted diamond labeled M is the membranous portion of the septum lying beneath the commissure between the right aortic cusp (*R*) and posterior aortic cusp (*P*). Adjacent is the anterior mitral leaflet (*AM*) The posterior mitral leaflet (*PM*) also is shown

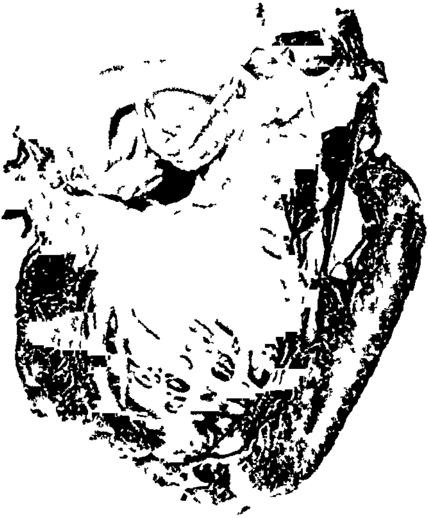


Fig 4 —Ventricular septal defect viewed from left ventricular aspect There is no septal tissue immediately beneath the commissure between the posterior and the right aortic cusps and these cusps overhang the defect.

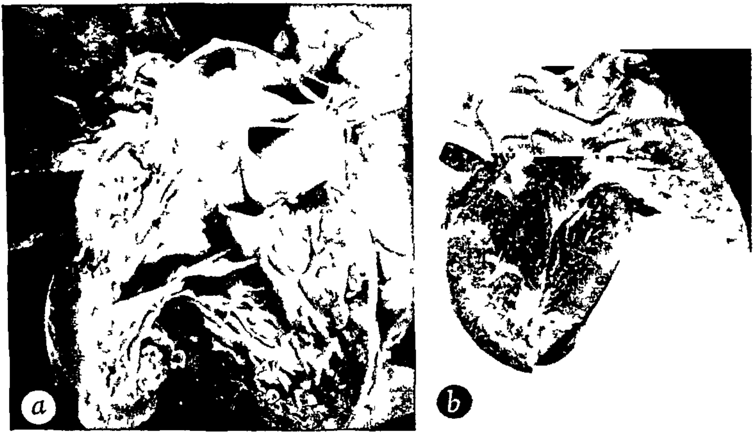


Fig 5 —Ventricular septal defect anterior to crista supraventricularis viewed from right ventricular (*a*) and left ventricular (*b*) aspect The defect is immediately beneath the pulmonary valve and just beneath the left aortic cusp

defect is placed essentially as it is in normally formed hearts, as described by Widran and Lev.[7] Thus there may be a close relationship of the bundle of His and its right and left branches to those defects that appear in the outflow tract of the right ventricle inferior to the crista supraventricularis. In such defects, the main bundle would be located in the tissue forming the postero-inferior extremity of the defect while the beginning of the right branch of the bundle of His would lie more or less parallel to the inferior edge of the ventricular septal defect, as the right branch runs forward toward the base of the papillary muscle of the conus. Beyond this, it turns downward along the ventricular septum at some distance from the position of the usual type of ventricular septal defect. The region of the base of the papillary muscle of the conus, while grossly related to the position of the right branch, is beyond the location of the origin of fibers of the left branch. The fibers of the left bundle in the normal heart are so placed as to cross toward the left in the tissue inferior to the position of the type of defects under discussion.

The atrioventricular node lying above the base of the septal leaflet of the tricuspid valve and in the lowermost part of the atrial septum at a position anterior to the orifice of the coronary sinus is not as intimately related to the position of ventricular septal defects as are the main bundle of His and its right and left branches.

Defects in the outflow tract of the left ventricle that are superior to the crista supraventricularis and immediately inferior to the pulmonary valve are not closely related to the position of the bundle of His and its branches. Defects in the apex of the septum are not intimately related to the position of the conduction system. Those defects lying beneath the septal leaflet of the tricuspid valve may be related to the position of elements of the right branch of the bundle of His.

### MATERIAL

In this presentation, no mention will be made of cases of a ventricular septal defect that is part of the tetralogy of Fallot since the technical considerations are somewhat different in these instances. Cases of common atrioventricular canal are likewise excluded.

Thirty-six patients who underwent surgical treatment of a ventricular septal defect between March 1, 1955, and April 15, 1956, comprise the basis for this report. The most commonly encountered type of defect was that related to the ventricular outflow tracts, inferior to the crista supraventricularis (see Table I). Nineteen of the twenty-five defects of this type seemed to be completely above the chordae tendineae to the papillary muscle of the conus while six were both above and below these chordae. In 12 of the 25 cases, the aortic leaflets could be seen prominently in the operative field as they overhung the defect. This indicates very little septal tissue, if any, remaining between the annulus of the aortic valve and the defect. In 10 cases, there was no posterior rim of residual membranous septum and the posterior border of the defect was formed by a membrane representing the continuous portion of the septal leaflet of the tricuspid valve and the anterior mitral leaflet.

There were associated defects in some cases (Table II). Associated atrial septal defect, patent ductus arteriosus, and mitral stenosis were corrected at the time the ventricular septal defect was repaired.

Table II. Associated Anatomic Defects in 36 Surgical Cases of Ventricular Septal Defect

| ASSOCIATED DEFECT | CASES |
|---|---|
| Atrial septal defect | 4 |
| Persistent left superior vena cava | 3 |
| Patent ductus arteriosus | 1 |
| Congenital mitral stenosis | 1 |
| Right aortic arch | 1 |

## COMPLETENESS OF REPAIR

Since many of the patients were small children, it has been difficult to urge early postoperative cardiac catheterization upon them. Even postoperative catheterization is not an infallible method for identifying small residual shunts unless done with extreme care and accuracy. In evidence of this is the fact that adult patients who have not been operated on and who have very loud systolic murmurs characteristic of maladie de Roger may have a left to right shunt so small as to be detectable only with difficulty by the usual catheterization techniques.

All would agree that complete absence of the systolic murmur of ventricular septal defect is excellent evidence of complete repair. The presence of a short, rough, Grade 1 or 2 systolic murmur over the precordium after operation does not prove the presence of a residual shunt, but the presence of such a murmur does not allow the patient to be considered as having a proved complete repair until the result is confirmed by cardiac catheterization. A patient with such a murmur but with a normal indicator dilution curve as determined in the operating room after repair, a quiet heart and clinical improvement as manifested by rapid growth, gain in weight and disappearance of symptoms presumably has at least a satisfactory repair and perhaps a complete repair. A persisting loud systolic murmur, combined with an indicator dilution curve determined immediately after repair and indicating persisting left to right shunt even though small, gives clear evidence of a residual shunt. When death occurs postoperatively, necropsy allows clear decision as to completeness of repair.

Table III. Results of Repair of Ventricular Septal Defect in the Entire Series of 36 Cases in Which Various Techniques Were Used and in the Last 25 Cases, in Which the Presently Employed Technique Was Used

| RESULT | VARIOUS TECHNIQUES (ALL 36 CASES) | PRESENT TECHNIQUE (LAST 25 CASES) |
|---|---|---|
| Complete repair | 20 | 18 |
| Satisfactory repair (may well be complete but not yet so proved) | 7 | 4 |
| Known residual shunt | 9 | 3 |

The completeness of repair for the present series as adjudged by these criteria is indicated in Table III. Techniques used include closure by direct suture, by a now abandoned technique using compressed polyvinyl formal

(Ivalon) sponge, and by the presently employed method. The last technique has been developed because of disappointment with occasional incomplete repair by the methods used earlier. That this technique, to be described, has been a distinct advance is indicated by the data portrayed in Table III; with it there have been only three instances of known residual shunts. One was in a patient with multiple ventricular septal defects, a very small one of which was closed imperfectly by a single stitch which has allowed a small residual shunt.

This experience emphasizes the importance of meticulous technique in the actual repair of these defects, made difficult in some instances by proximity of valvular structures and by problems of exposure of certain areas of the lesion. These obstacles must be overcome if the goal of complete, secure closure for each patient is to be reached.

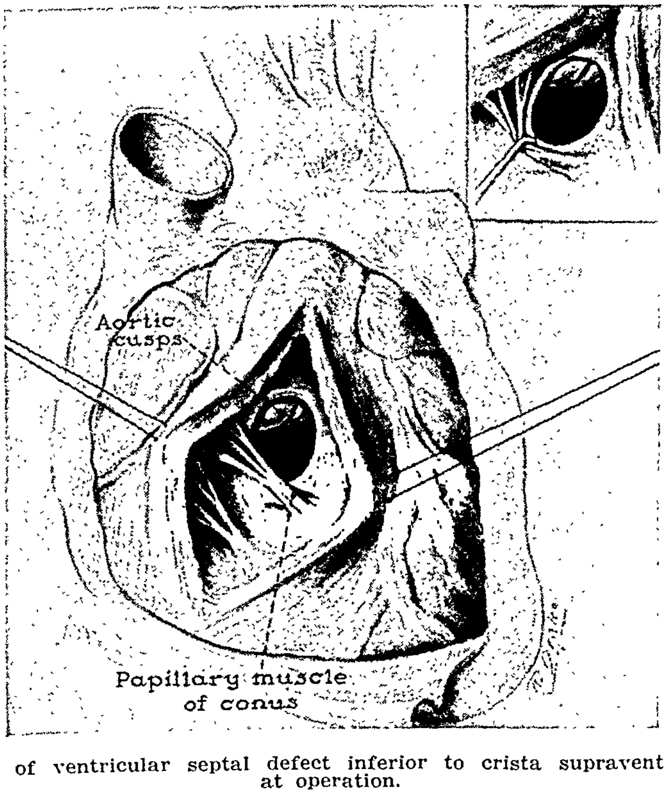


Fig. 9.—Drawing of ventricular septal defect inferior to crista supraventricularis as seen at operation.

## TECHNIQUE OF REPAIR

Since most defects seen surgically are related to the ventricular outflow tracts and are postero-inferior to the crista supraventricularis, closure of this type of defect will be described. Such defects are more difficult of secure repair than are other types. The technique described has been used in the last 25 cases of this series.

After the function of pumping and oxygenation of blood is taken over by the pump-oxygenator unit, the right ventricle is opened widely. The ventricular septal defect is identified and its relation to the aortic and tricuspid leaflets noted (Fig. 9). The postero-inferior area is scrutinized to determine whether this is composed of a remnant of membranous septum or whether the remnant is absent and instead the leaflets of the mitral and tricuspid valves are continuous across this area. The proposed placement of stitches is considered so as to avoid placing them directly posterior in a position which seems to invite conduction disturbances Should ventricular asystole or heart block occur as a stitch is being placed, warning has been served that this stitch should be removed and not tied down.

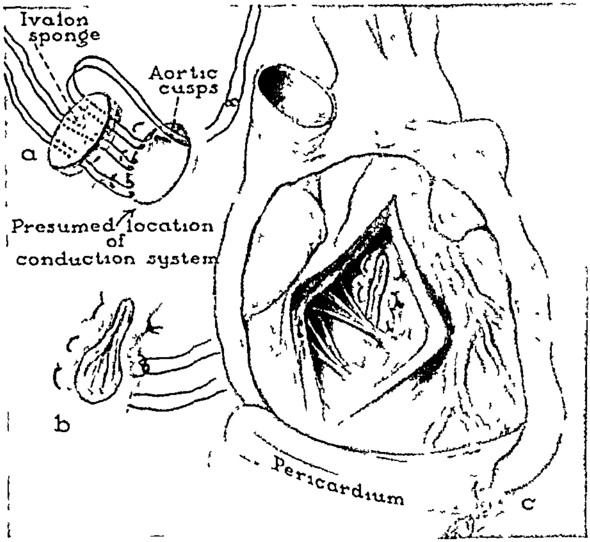


Fig 10—*a*, *b*, and *c*, Repair of ventricular septal defect using noncompressed polyvinyl-formal sponge Note in *a* the proximity of aortic cusps to the stitches Misplaced deep stitches in this area may tear an aortic cusp The area probably occupied by the conduction system should be avoided in stitching

On occasion, chordae tendineae attaching to the papillary muscle of the conus partly overlie these defects In such instances, they are retracted. Mattress sutures of 2 0 silk are then placed for repair so that they grasp one side of the defect, pass through an appropriately trimmed piece of noncompressed polyvinyl-formal (Ivalon) sponge and then through the other edge of the defect (Fig. 10) As the mattress stitches are tied down, they bring the edges of the defect together against the interposed sponge to obtain a secure closure The line of closure is parallel to the direction of outflow from the right ventricle but is at right angles to the direction of outflow from the left ventricle The opportunity for distortion of aortic valve leaflets is minimized because of this.

Sometimes the placement of these sutures in the inferior aspect of the right edge of the defect, as viewed from the right ventricular side, is difficult. This area may be formed by the tricuspid valve ring itself and is partially overhung by the septal leaflet of this valve. The most inferior pair of mattress sutures may, in such instances, be inserted into this margin of the defect through the tricuspid ring, penetrating it from the right atrial side. These sutures then go through the sponge and left edge of the defect to be tied down. If the defect lies completely beneath the septal leaflet of the tricuspid valve, it is often best to place all the mattress sutures into the right margin of the defect by suturing through the tricuspid valve ring (which forms this margin of such defects).

After the mattress sutures have been tied down, the repair is carefully scrutinized for residual tiny defects. If present, these must be securely closed by additional stitches. Likewise, after the obvious large defect is closed, the septum should be examined for smaller defects elsewhere. Bronchial blood coming to the left side of the heart will tend to appear through these small additional defects after the large one has been blocked. Such defects are closed by direct suture.

## COMMENT

The use of noncompressed polyvinyl-formal sponge in the manner described has been exceedingly satisfactory. The technique must be slightly altered for other types of "high" ventricular septal defects, although the basic principles are the same.

Direct suture of the "high" defects was employed in the first 3 of the 36 cases and in one later case which seemed ideally suited to it. Perhaps direct suture could be used more often than it was employed in this series but experience with the third case in which it was used discouraged its frequent application. In this case, the patient died eight hours after operation and necropsy disclosed that the suture line had partially disrupted, making the repair very incomplete. This, plus other experiences in the operating room, has lent conviction to the belief that the living ventricular septum allows tight stitches to cut through rather readily. In these large lesions, the use of a securely anchored polyvinyl-formed sponge to help fill the tissue defect allows a snug repair without the stitches having to be drawn too tightly.

## SUMMARY

A thorough understanding of the surgical anatomy of ventricular septal defect is essential to the successful treatment of it. Particular attention must be given to the variation in location of the defects. Exact knowledge of the relation of the defect to aortic leaflets, mitral and tricuspid leaflets, and the bundle of His is of inestimable value. The technique that has proved most satisfactory to date is described. Emphasis is placed on the necessity for precision in the repair to avoid damaging adjacent structures and to ensure absolute and permanent closure.

## ADDENDUM

Further experience since submission of this manuscript has caused the authors to adopt the routine use of potassium induced cardiac asystole in the repair of ventricular septal defects It is to be pointed out that the details of the actual closure with this technique are somewhat different than those described in this paper This is noted in an attempt to avoid misleading those interested in this field as to the method of repair now being employed by us

## REFERENCES

1 Jones, R E, Donald, D E, Swan, H J C, Harshbarger, H G, Kirklin, J W, and Wood, E H Apparatus of the Gibbon Type for Mechanical Bypass of the Heart and Lungs Preliminary Report, Proc Staff Meet, Mayo Clin 30 105 113, 1955
2 Harshbarger, H G, Donald, D E, and Kirklin, J W Studies in Extracorporeal Circulation III The Technic in Man for Open Intracardiac Surgery Unpublished data
3 Donald, D E, Harshbarger, H G, and Kirklin, J W Studies in Extracorporeal Circulation II A Method for the Recovery and Use of Blood From the Open Heart During Extracorporeal Circulation in Man, Ann Surg 144: 223 227, 1956
4 Kirklin, J W, Donald, D E, Harshbarger, H G, Hetzel, P S, Patrick, R T, Swan, H J C, and Wood, E H Studies in Extracorporeal Circulation I Applicability of Gibbon Type Pump Oxygenator to Human Intracardiac Surgery 40 Cases, Ann Surg 144 2 8 1956
5 DuShane, J W, Kirklin, J W, Patrick, R T, Donald, D E, Terry, H R, Jr, Burchell, H B, and Wood, E H Ventricular Septal Defects With Pulmonary Hypertension Surgical Treatment by Means of a Mechanical Pump Oxygenator, J A M A 160 950 953, 1956
6 Becu, L M, Fontana, R S, DuShane, J W, Kirklin, J W, Burchell, H B, and Edwards, J E Anatomic and Pathologic Studies in Ventricular Septal Defect, Circulation 14 349 364, 1956
7 Widran, J, and Lev, M The Dissection of the Atrioventricular Node, Bundle and Bundle Branches in the Human Heart, Circulation 4 863 867, 1951

## DISCUSSION

DR C W LILLEHEI, Minneapolis, Minn—I rise to heartily endorse the very fine presentation of Dr Kirklin, and to comment upon one of the ancillary problems related to closing septal defects that was not touched upon by either Dr Kirklin or Dr Warden due to time limitations I refer to the occurrence and persistence of complete heart block in occasional patients following intracardiac surgery I think it would be presumptuous to say that this problem has been solved, but we have learned a good deal about its management by the empiricism of trial and error and, since many more surgeons will be undertaking these procedures in the near future, it may be of value to summarize briefly some of the information of value that has been obtained by this painfully slow method of inquiry

The incidence of complete heart block has been about 12 per cent in the patients having ventricular septal defects repaired

There are probably two important elements inherent in the causation of this complication, one is the placement of the stitches so as to injure or interrupt the main conduction pathways I'll say a further word about that in a moment The other element is a little more difficult to define precisely, but is related to the status of the patient's cardiac reserve That is, among the patients having ventricular septal defects closed, there have been 11 cases of complete heart block and all have occurred in the patients with severe pulmonary hypertension, that is, in the group where the pulmonary pressures exceeded 70 to 80 per cent of the systemic pressures Thus, there is an increased predisposition to block where the myocardial reserve is less In fact, in an occasional instance in such a patient, we have seen complete heart block occur before any stitches had been placed in the heart The same consideration applies to the patients with tetralogy of Fallot, in those in whom the myocardium is severely hypoxic or the cardiac reserve is lessened greatly, heart block is more likely to occur

The conduction tissue has a high glycogen content and the pathways may be clearly demonstrated in the fresh heart by painting the septum with Lugol's solution.

(slide) These color photographs, prepared by Dr. Rivkin of our staff, show the anatomy of the conduction pathways in a fresh canine heart stained in this fashion. The atrioventricular node is shown here at the base of the interatrial septum lightly craniad to the coronary sinus. The atrioventricular bundle is seen as it proceeds horizontally forward, reaching the inferior edge of the membranous portion of the interventricular septum where it divides into left and right branches (bundle of His). Note in this photograph how quickly the left bundle, as it descends toward the apex, fans out into a diffuse broad sheet of fibers lying subendocardially. This diffuseness is a favorable anatomic circumstance for the surgeon since it makes it less likely that a single stitch will encompass the entire left bundle.

In this photograph of the right side of the septum in the same heart, the right bundle branch is seen as a continuation of the atrioventricular bundle. It passes undivided, subendocardially, to the anterior papillary muscle at which point it commences to branch out over the right ventricle.

Now, in the typically located ventricular defect due to absence of the membranous septum, this inferior margin of the membranous defect (apex of the muscular portion of the septum near the anterior margin of the tricuspid septal leaflet) is the most vulnerable area since a single stitch deeply placed here may encompass both right and left bundles causing complete heart block. In this area, we prefer placing our closure stitches on the right ventricular aspect of the defect margin, parallel with the defect margins, and not too deeply (so as not to penetrate to the left side). Thus, we often get a right bundle branch block, but this is innocuous since the rate and rhythm of the heart remain unchanged. If complete block is seen to occur with placement of a particular stitch, we generally remove the offending stitch and replace it in a slightly different place. This maneuver may or may not be of value but if it is not we continue with the closure stitches regardless, since our primary objective, if the patient is to survive, must be to close the defect.

The treatment that has proved lifesaving in those patients, in whom complete block persists, has been constant vigilance together with the use of the drug Isuprel (isopropyl levoarterenol). Death in patients with complete block occurs because the rate falls so low that the systemic pressure and coronary circulation deteriorate due to the diminished output, and cardiac arrest occurs. Successful treatment requires keeping the heart rate above this critical level (about 80 to 100 per minute in surgical patients) for a sufficient time interval until sinus rhythm recurs or the circulatory system has had a chance to readjust to the slower rate. In our first 3 patients we used epinephrine, ephedrine, atropine, and lactate for this purpose. They all produce some temporary beneficial effects, but all 3 patients eventually died due to their block.

We were a little slow to try Isuprel because an intravenous preparation was not available commercially. However, it is available now from Winthrop Stearns Company. We use a 1:50,000 solution and give 0.5 to 1.5 c.c. intravenously to infants, and up to 2 and 3 c.c. to adults per dose. With this dose the heart rate will accelerate and the effect often lasts for fifteen or twenty minutes. In some cases, there may actually be a sinus rhythm which may persist for a variable period, but often relapses again into complete block. In other cases, Isuprel merely keeps the heart rate at an acceptable level, somewhere above 90 to 100 beats per minute. Three or four hours after surgery, when the patient's condition has stabilized somewhat and the heart block has still persisted, we usually switch to the use of Isuprel linguets, 5 to 15 mg., given rectally. This form of administration is more satisfactory for long-term therapy inasmuch as an effect is produced within about thirty minutes which lasts from two to four hours. In all cases these patients must be monitored constantly by electrocardiography. If sinus rhythm recurs, as it has in all of our survivors, Isuprel should be continued in gradually diminishing quantities for another ten to fourteen days to prevent sudden deaths due to recurrence of complete block. Occasionally, Isuprel has appeared to lose its effectiveness, and this has been restored by combining the Isuprel with relatively small doses of intravenous molar lactate. Utilizing Isuprel, 5 of the last 8 patients developing complete heart block after closure of their ventricular defect have been saved, and all have recovered a sinus rhythm before leaving the hospital.

DR. ALFRED BLALOCK, Baltimore, Md.—Dr. Edward Churchill said at a recent meeting, when called upon to talk, that Senior Members should be seen but not heard. I cannot resist the temptation to congratulate the people who have presented these papers. It is fascinating work, and I am sure that when you become as old as I am you will look back on this era of the beginning of intracardiac surgery with a great deal of pleasure. Certainly this meeting has been a most interesting one and I have listened very intently. In leaving for home I do not know quite which attack to prefer. I am inclined to think that, whereas magnificent results have been obtained by the group in Minneapolis, probably the Kirklin modification of the Gibbon apparatus is today the best procedure to follow. I would predict, however, that in ten years this would seem as outmoded as some of you regard some of the procedures done ten years ago.

I was interested to hear John Kirklin at the beginning of his paper call these "corrective procedures." I truly believe this to be a better term than that used in Minneapolis, "curative procedures." My particular interest is in the tetralogy of Fallot, some of which may be partially corrected by previous techniques. In speaking of that lesion alone at the moment, when there is a mortality, as I understand Dr. Varco reported in New York recently, of 45 per cent, and when the patients have been followed for very short periods, I do not think—and I will admit to being old fashioned—that this should as yet be called a curative procedure. I do not wish to indicate that I am not fascinated and thrilled by these results which have been reported not only this afternoon but throughout the meeting. I might say, however, that developments are going so fast that the faster I run, the further I get behind.

DR. JOHN W. KIRKLIN (Closing).—I am sure that everyone in this audience is indebted to Dr. Blalock for his comments on this problem. Certainly those of us who are young and enthusiastic do not at times have the privilege of looking at things in their proper perspective.

I would like to second what Dr. Lillehei has said about the conduction system. In my anxiety and nervousness I forgot to point out something on the slide.

(slide) Dr. Lillehei has already mentioned this, but just to emphasize the point which we both feel is very important: in this inferior angle is the presumed location of the conduction system. Certainly we feel, although the evidence is perhaps not conclusive, that one should not put a stitch in that area. I think quoting results as evidence is always open to considerable question, but for what it is worth in the last 25 cases closed by this repair, we have had no heart block. In the cases done prior to that, 10 or 11, we had three heart blocks. One patient died on the sixth postoperative day. The other 2 survived with the Isuprel technique mentioned and resumed a normal sinus rhythm. Certainly it is a complication that we would all like to avoid. Thinking of those methods employing purposeful cardiac arrest, I suppose one question that must be asked when one is employing this technique is whether the advantages outweigh the disadvantage of not knowing when to withdraw a stitch should block occur.

Dr. Gerbode asked me to say a word about common atrioventricular canal. That of course is a totally different type of defect with regard to technical considerations. It is an operable defect. We have had the opportunity to operate on 9 of these patients and 6 of them have survived operation; we have lost 3. We cannot really say for certain but, for what it is worth, it seems to me that this is the place where I would almost be willing to say it is absolutely necessary to use a prosthesis. There is a large tissue defect and it would seem that repair by direct suture must always be under considerable tension, and one wonders if that does not predispose in some way to heart block.

# THE USE OF THE HEART-LUNG APPARATUS IN HUMAN CARDIAC SURGERY

F. D. DODRILL, M.D., NORMAN MARSHALL, M.D. (BY INVITATION), JAN NYBOER, M.D. (BY INVITATION), C. H. HUGHES, B.S. (MED. BIOL.) (BY INVITATION), A. J. DERBYSHIRE, PH.D. (BY INVITATION), AND A. B. STEARNS, M.D. (BY INVITATION)
DETROIT, MICH.

DIFFERENT types of heart-lung machines are now in clinical use.[1, 2] All these types of mechanical devices have as their function the removal of venous blood as it returns to the heart, its arterialization and return to the aortic system, thereby taking over the function of the heart and lungs. There are, at the present time, two points of view as to the method of by-passing the heart and lungs using the varius types of machines. First of these involves the circulation and arterialization of a portion of the total blood volume. This is referred to as the low flow principle in extracorporeal circulation.[3] The desirable amount of blood to circulate in the low flow principle has not been definitely decided upon and has varied from 20 c.c. per kilogram of body weight per minute up to 50 to 60 c.c. per kilogram of body weight per minute. The normal blood volume in man is approximately 78 to 84 c.c. per kilogram of body weight, being slightly higher for males than for females.[4] Patients with left-to-right intracardiac shunts have greater than normal blood volumes because of the great amount contained in the dilated pulmonary vessels. The second principle in the extracorporeal circulation is the circulation of the total blood volume. There are advantages and disadvantages to both plans in the extracorporeal circulation. This manuscript deals with the circulation of the total blood volume, its complete arterialization, and the maintenance of normal systolic and diastolic arterial pressures.

## LOW FLOW PRINCIPLE

One of the chief reasons why the low flow principle is being carried out in extracorporeal circulation is the fact that it has not been possible, heretofore, to handle the total blood volume, to completely arterialize it, and to maintain normal pressures. While this has been discouraging over the years, it seems more hopeful that this plan will become a practical procedure. The advantages of a low flow circulation are that this can be handled more easily, that the apparatus needs to be less formidable and, more importantly, that the injury to the blood may be less because the amount which is circulating outside the body at a given time is less. There seems to be no definite advantage in the low flow principle aside from these facts.

---

From the Research Division of Harper Hospital and the Research Laboratories Division, General Motors Corporation.

Aided by a grant from The Michigan Heart Association.

Read at the Thirty-sixth Annual Meeting of The American Association for Thoracic Surgery at Miami Beach, Fla., May 7 to 9, 1956.

There are obvious disadvantages to the low flow principle The circulating blood volume is less and, therefore, the oxygen which is transported to the tissues is definitely less This is the chief and most important disadvantage of the principle With low flows, the arterial pressures are lower than if normal flows are used While these disadvantages may not be so great as to lead to death, it would seem better if the subject could be maintained on normal physiologic standards In young patients, circulation, of course, can be shut off for short periods of time If one then circulates a small amount of blood which is properly arterialized this period of occlusion can be extended for varying periods of time It seems, therefore, that the use of the low flow principle, *as the goal,* in extracorporeal circulation is not satisfactory

While many successful operations on patients have been performed with open cardiac surgery maintaining substandard physiologic processes, it would seem logical to believe that, if normal physiologic values were maintained, the over all results would be superior

## TWO INNOVATIONS

For a description of the apparatus used, the reader is referred to our previous publications[5 6] However, two innovations have been made in the plan of the operative technique The first of these consists in a so called exchange transfusion in which the patient's blood is partially removed together with some donor blood, placed in a properly prepared flask beside the operating table while the patient is maintained on the remainder of his own blood together with donor blood At the conclusion of the pumping time, the used blood is washed out to a certain extent, while the original blood is returned to his system, following which the pump arterializer is disconnected from the system The second innovation is in the return of the blood to the aortic system This is accomplished by suturing onto the side of the descending aorta, just below the origin of the left subclavian artery, a homologous aortic graft of sufficient size and caliber to permit the return of the total circulation to the aorta without its having to go through a narrow orifice such as the left subclavian artery

*Exchange Transfusion*—

One of the major drawbacks to extracorporeal circulation has been the injury to the blood Some of these injuries are known while some are unknown It has been customary to think of the injury to the blood only in terms of hemolysis of the red cells Inasmuch as the injury to the blood may be due not only to the actual physical trauma but also to complex changes in the chemical and enzyme systems, the amount of hemolysis in the blood is one of the ancillary manifestations of the total over all alteration in the blood constituents One of the discouraging complications, therefore, in extra corporeal circulation has been the development of the bleeding diathesis following the operative procedure This bleeding diathesis may be prevented in different ways It may be possible to add certain substances to the blood which will prevent its alteration during the extracorporeal circulation Less traumatic machines may be constructed which will more nearly simulate the

# THE USE OF THE HEART-LUNG APPARATUS IN HUMAN CARDIAC SURGERY

F. D. DODRILL, M.D., NORMAN MARSHALL, M.D. (BY INVITATION), JAN NYBOER, M.D. (BY INVITATION), C. H. HUGHES, B.S. (MED. BIOL.) (BY INVITATION), A. J. DERBYSHIRE, PH.D. (BY INVITATION), AND A. B. STEARNS, M.D. (BY INVITATION)
DETROIT, MICH.

DIFFERENT types of heart-lung machines are now in clinical use.[1, 2] All these types of mechanical devices have as their function the removal of venous blood as it returns to the heart, its arterialization and return to the aortic system, thereby taking over the function of the heart and lungs. There are, at the present time, two points of view as to the method of by-passing the heart and lungs using the varius types of machines. First of these involves the circulation and arterialization of a portion of the total blood volume. This is referred to as the low flow principle in extracorporeal circulation.[3] The desirable amount of blood to circulate in the low flow principle has not been definitely decided upon and has varied from 20 c.c. per kilogram of body weight per minute up to 50 to 60 c.c. per kilogram of body weight per minute. The normal blood volume in man is approximately 78 to 84 c.c. per kilogram of body weight, being slightly higher for males than for females.[4] Patients with left-to-right intracardiac shunts have greater than normal blood volumes because of the great amount contained in the dilated pulmonary vessels. The second principle in the extracorporeal circulation is the circulation of the total blood volume. There are advantages and disadvantages to both plans in the extracorporeal circulation. This manuscript deals with the circulation of the total blood volume, its complete arterialization, and the maintenance of normal systolic and diastolic arterial pressures.

## LOW FLOW PRINCIPLE

One of the chief reasons why the low flow principle is being carried out in extracorporeal circulation is the fact that it has not been possible, heretofore, to handle the total blood volume, to completely arterialize it, and to maintain normal pressures. While this has been discouraging over the years, it seems more hopeful that this plan will become a practical procedure. The advantages of a low flow circulation are that this can be handled more easily, that the apparatus needs to be less formidable and, more importantly, that the injury to the blood may be less because the amount which is circulating outside the body at a given time is less. There seems to be no definite advantage in the low flow principle aside from these facts.

---

From the Research Division of Harper Hospital and the Research Laboratories Division, General Motors Corporation.

Aided by a grant from The Michigan Heart Association.

Read at the Thirty-sixth Annual Meeting of The American Association for Thoracic Surgery at Miami Beach, Fla., May 7 to 9, 1956.

There are obvious disadvantages to the low flow principle. The circulating blood volume is less and, therefore, the oxygen which is transported to the tissues is definitely less. This is the chief and most important disadvantage of the principle. With low flows, the arterial pressures are lower than if normal flows are used. While these disadvantages may not be so great as to lead to death, it would seem better if the subject could be maintained on normal physiologic standards. In young patients, circulation, of course, can be shut off for short periods of time. If one then circulates a small amount of blood which is properly arterialized, this period of occlusion can be extended for varying periods of time. It seems, therefore, that the use of the low flow principle, *as the goal,* in extracorporeal circulation is not satisfactory.

While many successful operations on patients have been performed with open cardiac surgery maintaining substandard physiologic processes, it would seem logical to believe that, if normal physiologic values were maintained, the over-all results would be superior.

### TWO INNOVATIONS

For a description of the apparatus used, the reader is referred to our previous publications.[5, 6] However, two innovations have been made in the plan of the operative technique. The first of these consists in a so-called exchange transfusion in which the patient's blood is partially removed together with some donor blood, placed in a properly prepared flask beside the operating table while the patient is maintained on the remainder of his own blood together with donor blood. At the conclusion of the pumping time, the used blood is washed out to a certain extent, while the original blood is returned to his system, following which the pump arterializer is disconnected from the system. The second innovation is in the return of the blood to the aortic system. This is accomplished by suturing onto the side of the descending aorta, just below the origin of the left subclavian artery, a homologous aortic graft of sufficient size and caliber to permit the return of the total circulation to the aorta without its having to go through a narrow orifice such as the left subclavian artery.

*Exchange Transfusion.—*

One of the major drawbacks to extracorporeal circulation has been the injury to the blood. Some of these injuries are known while some are unknown. It has been customary to think of the injury to the blood only in terms of hemolysis of the red cells. Inasmuch as the injury to the blood may be due not only to the actual physical trauma but also to complex changes in the chemical and enzyme systems, the amount of hemolysis in the blood is one of the ancillary manifestations of the total over-all alteration in the blood constituents. One of the discouraging complications, therefore, in extracorporeal circulation has been the development of the bleeding diathesis following the operative procedure. This bleeding diathesis may be prevented in different ways. It may be possible to add certain substances to the [illegible] which will prevent its alteration during the extracorporeal circulation. [illegible] traumatic machines may be cons[illegible] heart and vascular system. Finally, the injured and deleterious components of the blood may be removed, or partially removed, in a process of exchange transfusion similar to that which is performed in erythroblastosis fetalis. In this condition, the infant's blood is incompatible with its own system. The noxious elements may be partially removed by process of exchange transfusion whereby a quantity of the patient's own blood is removed and replaced with compatible donor blood. It is not necessary or possible to remove all the patient's blood in this condition but, rather, the noxious elements are diluted down to the point where they are sublethal.

When one operates on a small patient who has a blood volume of 500 to 1,000 c.c. of blood, there is almost complete removal of his blood in the extracorporeal circuit. At least, one removes a large part of the patient's blood and replaces it with mixed donor blood. While this can be done effectively in most cases, it is probable that certain complications in cross matching and incompatible blood might possibly enter into the picture. On the other hand, if a fairly good portion of his own blood is preserved and returned to his circuit at the end of the operation, these possible complications might be obviated.

At the conclusion of any pumping period, it is probable that the serum potassium is elevated in all cases. This degree of elevation is not necessarily parallel, or dependent upon, the amount of hemolysis in the blood. This can be greatly reduced by the exchange transfusion at the conclusion of the procedure.

While it may not be necessary for short runs, such as thirty to forty minutes, one never knows exactly how long will be required to repair the defect. Moreover, the time is now at hand when more complex defects may be repairable. The fact of the matter is that there are no nontraumatic heart-lung machines available at the present time.

When we are ready to place the patient on the pump arterializer, he is given heparin solution in the amount of 4 mg. per kilogram of body weight. At the conclusion of the procedure and after the cannulae are removed from the patient, he is given protamine sulfate in the proportion of 1.2 to 1 of the amount of heparin. The clotting mechanism has returned to normal in all cases within forty-five minutes from the time the protamine has been administered. In these patients who have been carefully studied as to their clotting times postoperatively, we have seldom observed the so-called rebound phenomenon, that is, having the clotting times repeatedly return to normal but in a few hours rise again to a prolonged clotting time. The actual explanation for this rebound phenomenon has not been forthcoming but we suggest that a possible explanation of this is the postoperative breakup of the red cells which, in itself, may release additional heparin.

The advantages of this exchange transfusion are so great and so protean that time and space do not permit a complete statement of its advantages. Suffice it to say, that the advantages are great and that the possibilities for good have not been completely utilized. Moreover, aside from the mechanical heart-lung work, this may be of some use in other forms of clinical surgery in which uncontrolled bleeding from unknown causes ensue.

It has occurred to us, therefore, that since complex alterations may be taking place in the blood during extracorporeal circulation which may not be fully understood at the present time, to remove the noxious elements by performing a so-called exchange transfusion. With the pump arterializer connected to the patient, the technical aspect of the procedure is extremely simple.

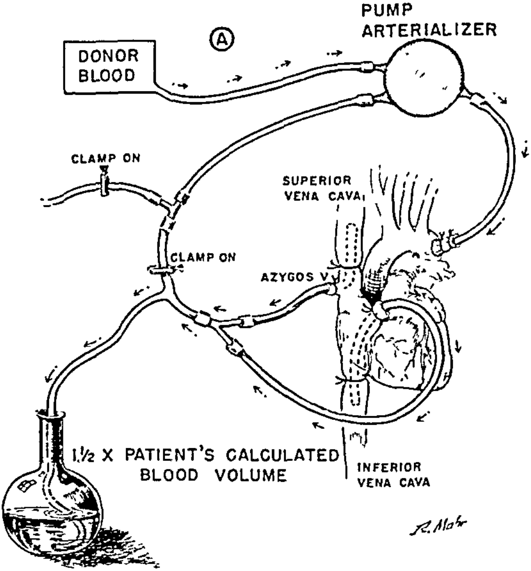
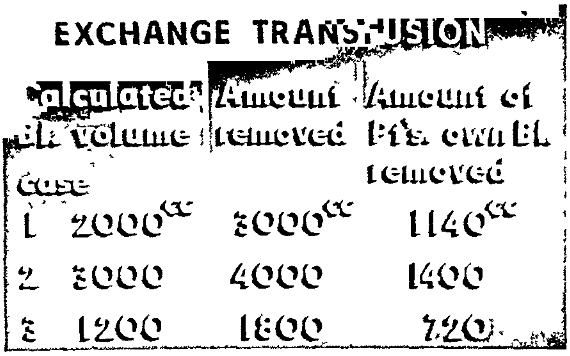


Fig. 1.—A diagrammatic illustration of the technique of removing the patient's blood

*Technique of exchange transfusion:* With the pump arterializer connected to the patient as for extracorporeal circulation, the apparatus is turned on and blood is allowed to enter the aorta under normal pressure while the venae cavae blood is allowed to collect in a properly prepared vessel by the side of the operating table (Fig. 1). Normal aortic arterial pressures must be maintained in order that the volume of blood put into the aorta may be equal to that which is removed from the venae cavae. It is amazing that under these circumstances the volume of blood which is collected by the side of the operating table practically always equals that which is put into the arterial system. Obviously, there is mixing of the donor and patient's blood during the procedure. It has been our practice to remove one and one-half times the calculated volume of the patient's blood. This is an arbitrary figure but has been found to be satisfactory in the amount of the patient's actual blood removed. Using radioactive serum albumin for blood volume determinations, it has been found that the removal of one and one-half times the patient's volume results in the removal of from 35 to 40 per cent of his own actual blood

(Fig. 2). At the conclusion of the extracorporeal circulation, this volume of blood is passed through the pump arterializer one time and returned to the patient's circulation following which the pump is disconnected from the vascular system (Fig. 3).



EXCHANGE TRANSFUSION

| Case | Calculated Bl. volume | Amount removed | Amount of Pt's own Bl. removed |
|---|---|---|---|
| 1 | 2000cc | 3000cc | 1140cc |
| 2 | 3000 | 4000 | 1400 |
| 3 | 1200 | 1800 | 720 |

Fig. 2.—Blood volume studies on 3 patients. Radioactive isotope technique using iodinated human serum albumin.

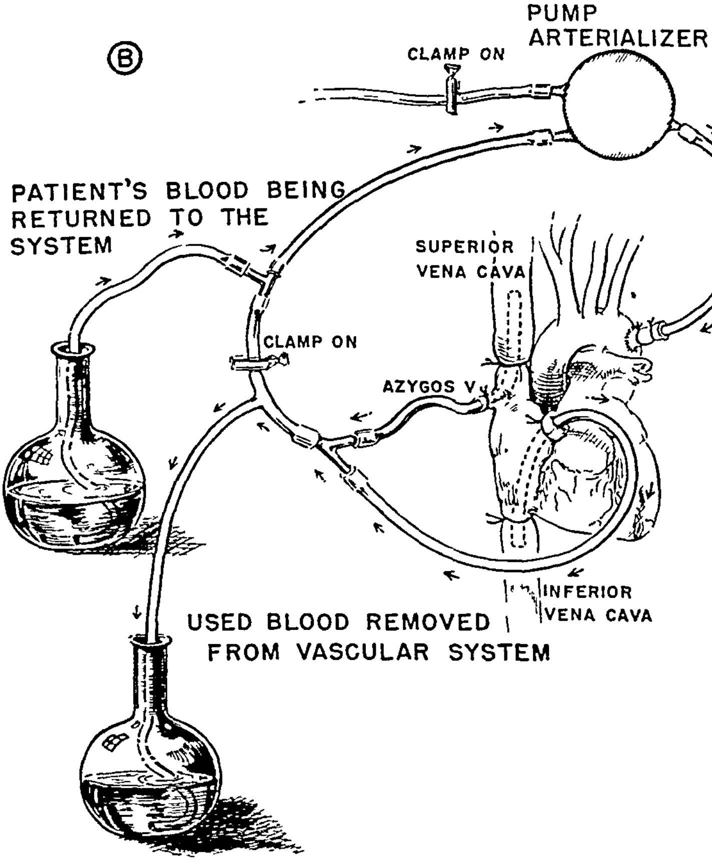

Ⓑ
PUMP ARTERIALIZER
CLAMP ON
PATIENT'S BLOOD BEING RETURNED TO THE SYSTEM
SUPERIOR VENA CAVA
CLAMP ON
AZYGOS V.
INFERIOR VENA CAVA
USED BLOOD REMOVED FROM VASCULAR SYSTEM

Fig. 3.—Diagrammatic illustration of the technique of returning the patient's own blood to his vascular system and the simultaneous removal of the used blood.

*Return of the Blood to the Aorta.*—

It is our belief, based upon several years' experience with extracorporeal circulation, that it is not possible to maintain good arterial pressures by returning the subject's blood volume by way of the subclavian artery. This is obviously true when one considers that the orifice of the subclavian artery may be only from one third to one fourth the actual size of the aortic valve through which the blood volume has been ejected in the subject. Also, the return of the blood to the aortic system by way of the small subclavian orifice is obviously one of the major points in the turbulence of the blood. Moreover, the return of the blood through a small orifice dissipates the pulse pressure to a large degree. While it has not been definitely shown that a pulse pressure is necessary, we believe that it is desirable to maintain it, if possible. Likewise, the maintenance of normal arterial pressure both systolic and diastolic during the extracorporeal circulation has not been proved to be absolutely necessary to life for short periods. While many patients throughout the country have been successfully operated upon with good recovery with a low arterial pressure during the extracorporeal circulation, the desirable thing to do is to maintain normal systolic and diastolic pressures according to our normal standard physiologic values. This can be accomplished only by the return of the blood to the aorta through a larger orifice than the left subclavian artery, in fact, the orifice should be essentially and nearly equal to the aortic valve of the particular subject being operated upon.

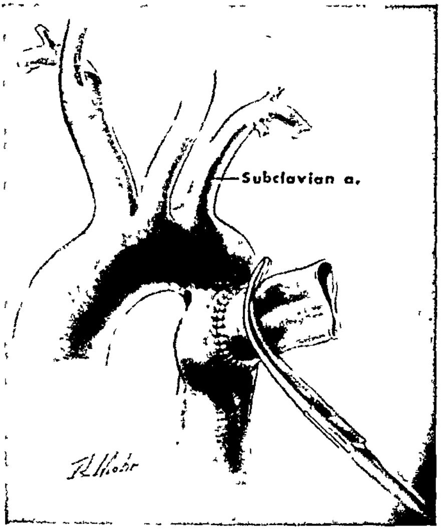


Fig 4—Diagrammatic illustration of an aortic graft sutured onto the side of the descending aorta

We are, therefore, making a linear incision on the lateral surface of the aorta just below the origin of the left subclavian artery, suturing onto this an arterial graft of proper size and caliber (Fig. 4). This orifice is usually made as large as the approximate diameter of the aorta itself.

fact that this adds something to the operative procedure but we are willing to accept this disadvantage for the obvious advantages one obtains in having complete control over the extracorporeal circulation once it is connected. This is our first attempt to return the blood through a large orifice. There may be more simple and practical ways of doing it than the suturing on of a graft on the side of the aorta.

## APPLICATION TO PATIENTS

We have applied this new technique to a group of 6 patients. The types of anatomic defects operated upon have all been interventricular septal defects with the exception of one patient with tetralogy of Fallot, who incidentally survived. They have been maintained on normal physiologic standards insofar as we have been able to determine. These physiologic checks include the electrocardiogram, electroencephalogram, intra-arterial and central venous pressures together with the usual blood checking data such as arterial oxygen saturation, plasma carbon dioxide, and pH. The pumping period has varied from twenty-one minutes to fifty-two minutes' time. The average has been twenty-five to thirty minutes. The prolonged pumping period in the one patient resulted because of the fact that, after the ventricular septal defect in the high membranous septum had been closed by method of suture, there was a rupture in a separate portion of the septum aside from the suture line and a large defect resulted. This required an additional thirty-five minutes to repair. In some of the instances, the physiologic data have been more normal while the patient was on the pump arterializer than immediately before he was put on it.

In these six attempts, using this method, 3 patients have died postoperatively giving us an operative mortality of 50 per cent. Two of these patients were operated upon earlier when the method of inflating the lungs with helium was not currently practiced and both of these patients succumbed to pulmonary atelectasis (Fig. 5). A third patient died from intrapericardial hemorrhage forty-eight hours, postoperatively. All of these patients have temporarily recovered from the procedure and have lived at least forty-eight hours.

## VOLUME FLOWS

Using this plan of returning the blood to the aorta through a large orifice, we are able to maintain perfectly normal blood flows. It is our experience that in patients with congenital heart disease with a left-to-right shunt that the total circulating blood volume is in excess of normal values. In all of the cases to date, it has been found that the circulating volume has been from 90 to 100 c.c. per kilogram of body weight per minute using radioactive albumin. This has actually been the blood flow in all cases which has been determined by a Wier-type flowmeter.

One of the amazing phenomena or observations following this plan is that the amount of postoperative drainage is extremely small. Of the 6 patients, all of whom survived at least forty-eight hours postoperatively, in only

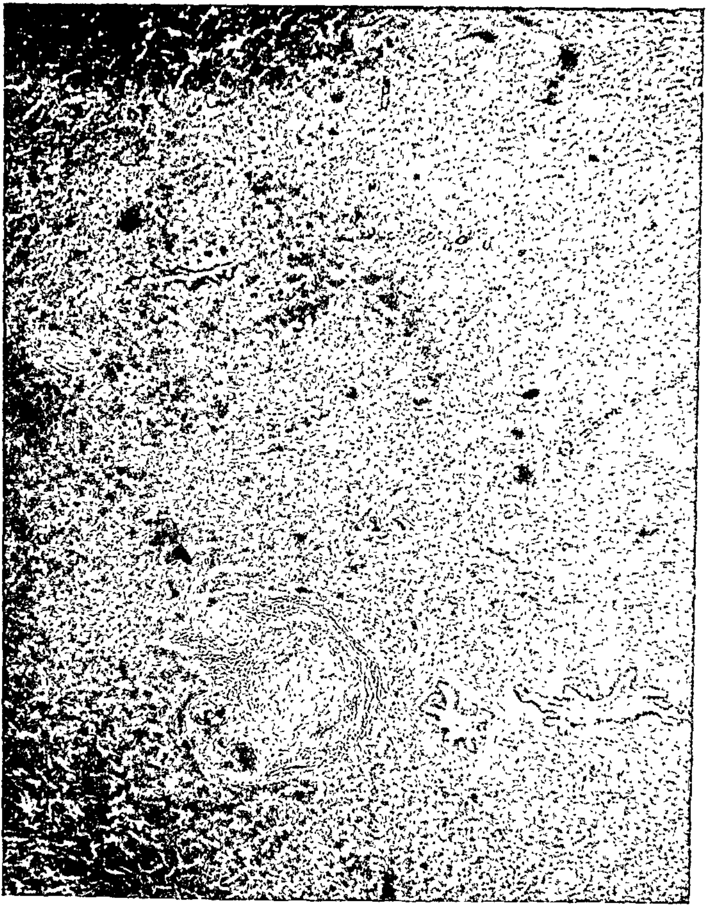


Fig. 5.—Microscopic section of postoperative atelectasis of the lung.

one was there a drainage of from 300 to 350 c.c. of serum. In all the others the drainage was so minimal that it was hardly measurable. This, in itself, indicates to us that the enzyme mechanisms of the blood are essentially normal.

### TYPE OF SUTURE USED IN REPAIR OF INTERVENTRICULAR SEPTAL DEFECT

Fig. 6 illustrates a figure-of-eight type of suture which was used to approximate the septal defect. While this may not be accepted by all as superior to a single interrupted suture, it is the impression that a figure-of-eight stitch properly placed actually has less pressure upon a given point of the tissue than if a single interrupted one were used.

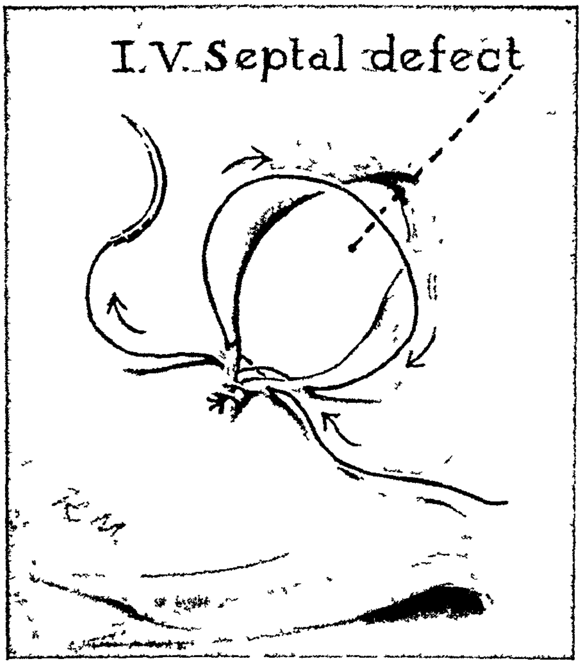


Fig. 6—Illustration of a figure-of-eight type of suture for repair of interventricular septal defects

### PHYSIOLOGIC DATA USING THE EXCHANGE TRANSFUSION AND THE LARGE ORIFICE IN THE AORTA FOR THE RETURN OF THE CIRCULATION

Fig. 7 illustrates the patient's blood presssure before, during the exchange transfusion, and after being completely on the pump arterializer. It is seen at a glance that the systolic and diastolic pressures both during the exchange transfusion and subsequent to it, during the pumping period, are entirely normal, being about 120/80 mm. Hg. It has been the experience of most investigators that during the pumping period the arterial pressure tends to drop somewhat during the period. It has been our experience, on the other hand, that, uniformly, the pressure rises as the pumping period continues and stabilizes near the preoperative level. This is illustrated in Fig. 8 One unphysiologic phenomenon which we have observed during this procedure is the rise in the venous pressure. This rise in venous pressure does not necessarily mean that too much blood is being pumped. It may be adequately explained

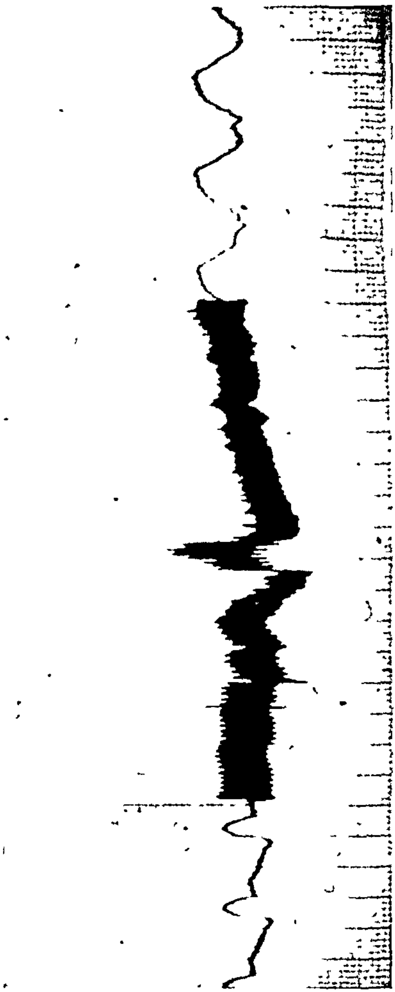


Fig 7.—The actual record of the arterial blood pressure before pumping, during the exchange transfusion, and after being completely on the pump arterializer

one was there a drainage of from 300 to 350 c.c. of serum. In all the ot
drainage was so minimal that it was hardly measurable. This, in itse
cates to us that the enzyme mechanisms of the blood are essentially no

### TYPE OF SUTURE USED IN REPAIR OF INTERVENTRICULAR SEPTAL DEFEC

Fig. 6 illustrates a figure-of-eight type of suture which was used
proximate the septal defect. While this may not be accepted by all
perior to a single interrupted suture, it is the impression that a figure-o
stitch properly placed actually has less pressure upon a given point
tissue than if a single interrupted one were used.

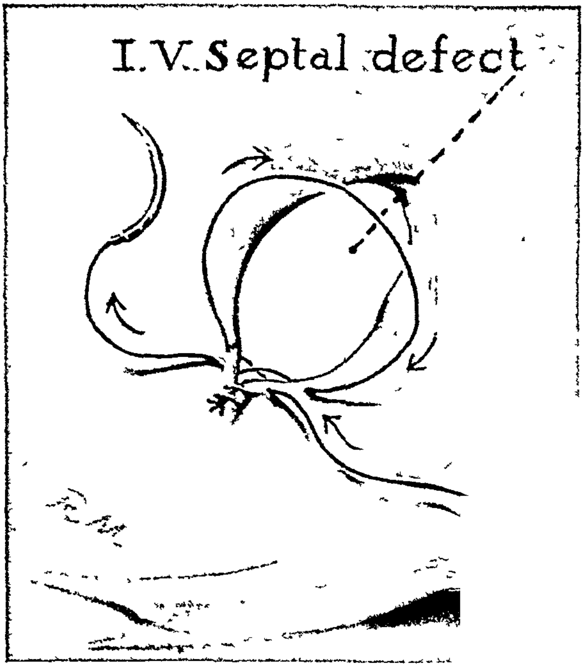
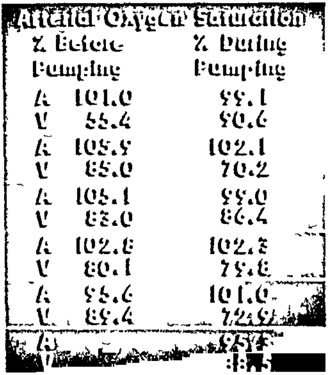


Fig. 6.—Illustration of a figure-of-eight type of suture for
defects.

### PHYSIOLOGIC DATA USING THE EXCHANGE TRANSF IN THE AORTA FOR THE RETURN OF

Fig. 7 illustrates the patient's blood pres
transfusion, and after being completely on
at a glance that the systolic and diastolic
transfusion and subsequent to it, durin
normal, being about 120/80 mm. Hg. I
vestigators that during the pumping pe
somewhat during the period. It has
that, uniformly, the pressure rises
stabilizes near the preoperative le
physiologic phenomenon which w
rise in the venous pressure. Th
mean that too much blood is

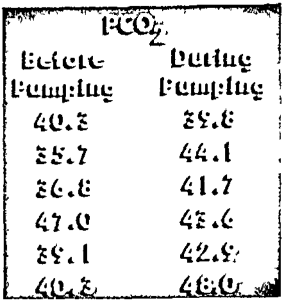
*The pH Values.*—During our previous investigative work over the years, it was our experience that during the pumping period the pH of the blood tended to drop below normal values. This has been thought of and has been spoken of as due to metabolic acidosis and was thought to be due to insufficient circulation. It is interesting to note that, with our plan of total extracorporeal circulation maintaining normal systolic and diastolic blood pressures, this fall in pH does not take place. Fig. 11 illustrates the pH taken during the pumping period and illustrates a perfectly normal value. To our experience, this can only be obtained by the maintenance of normal systolic and diastolic pressure and a maintenance of normal flow.



Arterial Oxygen Saturation

| | % Before Pumping | % During Pumping |
|---|---|---|
| A | 101.0 | 99.1 |
| V | 55.4 | 90.6 |
| A | 105.9 | 102.1 |
| V | 85.0 | 70.2 |
| A | 105.1 | 99.0 |
| V | 83.0 | 86.4 |
| A | 102.8 | 102.3 |
| V | 80.1 | 79.8 |
| A | 95.6 | 101.0 |
| V | 89.4 | 72.9 |
| A | [illegible] | [illegible] |

Fig 9.



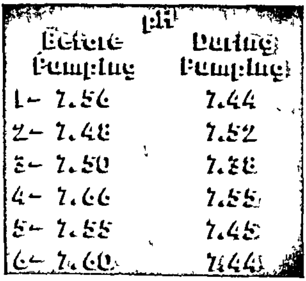
PCO₂

| Before Pumping | During Pumping |
|---|---|
| 40.3 | 39.8 |
| 35.7 | 44.1 |
| 36.8 | 41.7 |
| 47.0 | 43.6 |
| 39.1 | 42.9 |
| 40.3 | 48.0 |

Fig 10

Fig 9.—Oxygen saturation determinations by the Van Slyke method. Oxygen saturation of arterial and venous blood before pumping and toward the end of the pumping period in 6 patients

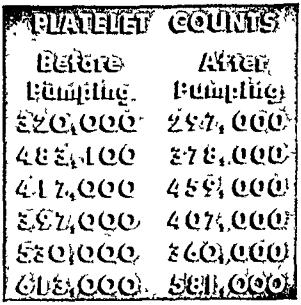
Fig 10.—$PCO_2$ determinations by the Van Slyke method $PCO_2$ before pumping and toward the end of the pumping period in 6 patients



pH

| | Before Pumping | During Pumping |
|---|---|---|
| 1- | 7.56 | 7.44 |
| 2- | 7.48 | 7.52 |
| 3- | 7.50 | 7.38 |
| 4- | 7.66 | 7.55 |
| 5- | 7.55 | 7.45 |
| 6- | 7.60 | 7.44 |

Fig 11.



PLATELET COUNTS

| Before Pumping | After Pumping |
|---|---|
| 320,000 | 297,000 |
| 483,100 | 378,000 |
| 417,000 | 459,000 |
| 397,000 | 407,000 |
| 530,000 | 360,000 |
| 618,000 | 581,000 |

Fig 12.

Fig 11.—The pH determinations by the Beckman pH meter. Before pumping and toward the end of the pumping period in 6 patients

Fig 12.—Platelet counts before and after extracorporeal circulation in 6 patients.

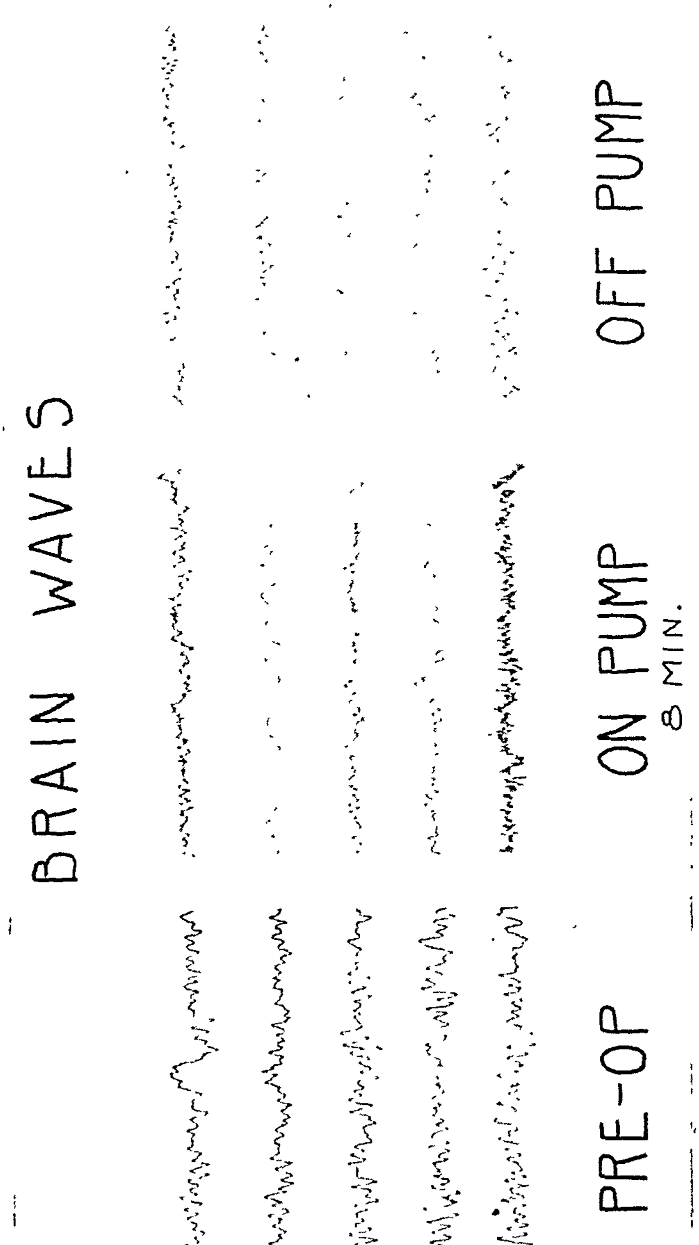


Fig. 13.—The electroencephalogram before, during pumping, and immediately after pumping.

*Blood Platelets*—The blood platelets were counted before and immediately after the pumping time No destruction is seen (Fig 12)

*Electroencephalogram*—The electroencephalogram was taken on all cases This was found to be normal except for a brief period, lasting two to three minutes during the exchange transfusion, when there was some abnormality (Fig 13)

## SUMMARY

Two innovations in extracorporeal circulation are presented The first involves the so called exchange transfusion in which a portion of the blood is removed and placed on the operating table while the patient is maintained on donor blood At the conclusion of the procedure, the original blood removed is returned to his system following which the pumping device is disconnected Indications are that the blood is essentially normal following this procedure

The second innovation involves the suturing of an aortic graft on the side of the aorta producing a large orifice through which the arterialized blood is returned to the system

Using this plan of procedure, the total blood volume of any subject can be properly arterialized and the systolic and diastolic arterial pressure maintained according to normal standards Indications from various physiologic data show that the total body physiology is likely to be more normal using this plan of circulation

By this procedure, 6 patients have been operated upon employing this technique During the procedure, various data have been recorded which include the arterial pressure central venous pressure, the effects on the electroencephalogram and the electrocardiogram Various blood chemistry data have been presented All data obtained indicate a more normal body physiology can be obtained than has heretofore been illustrated

## REFERENCES

1 Duchane, J W, Kirklin, J W, Patrick, R T, Donald, D E, Terry, H R, Jr, Burchell, H B and Wood, E H Ventricular Septal Defects With Pulmonary Hypertension J A M A 160 950 953, 1956
2 Lillehei, C W, and DeWall, R A American College of Surgeons, Postgraduate Cardiovascular Surgery Chicago, Ill, October, 1955
3 Cohen, M, and Lillehei, C W A Quantitative Study of the "Azygos Factor" During Vena Caval Occlusion in the Dog, Surg, Gynec & Obst 98 225 1954
4 Best and Taylor Physiological Basis of Medical Practice, ed 5, Baltimore, 1950, Williams & Wilkins Company, chapt 3, pp 16 31
5 Dodrill, F D Lui, A, Nyboer, J, Rippingille F V, and Hughes, C H The Arterialization of Blood as It Applies to the Mechanical Heart Lung Apparatus, J Thoracic Surg 30 658 664, 1955
6 Dodrill F D Hill, E and Gerisch, R A Some Physiologic Aspects of the Artificial Heart Problem, J Thoracic Surg 24 134, 1952

## DISCUSSION

DR JOHN H GIBBON, JR, Philadelphia, Pa—In the first place, I would like to say that, as Dr Dodrill well knows, I am in complete agreement with his point of view that we should attempt to have as nearly normal conditions as possible during the period of open intracardiac surgery, just as during any operative procedure In other words, we would like to avoid hemolysis of the blood, we would like to have the blood adequately saturated with oxygen, we would like to have a normal blood flow, and we would like to avoid either acidosis or alkalosis I could not be more heartily in favor with that pr tainly his figures of venous oxygen saturation would indicate that he had an adequate blood flow during the period of by-pass. I would be the first to agree that probably more things happen to the blood passing through an artificial circuit outside the body than the obvious things which can be detected by measurement, such as the breaking down of the cellular elements of the blood. Many of us have been studying the blood and have found great difficulty in putting our fingers on anything specific, except the fact that the leukocytes and platelets tend to decline in concentration. The effect upon the erythrocytes can be detected by studying the amount of free hemoglobin in the plasma.

I was sorry Dr. Dodrill did not indicate the level of free hemoglobin in the plasma of the donor blood. I presume it was reasonably high or he would not have employed an exchange transfusion. I take it also that the amounts of free hemoglobin in the plasma which he reported are the figures after replacement with the recipient's own blood, and not those of the donor blood which was used during the by-pass. Many of us have reported the levels of free hemoglobin in the plasma, the decline in white blood cell count, and the decline in platelet count. The decline in platelet count is not severe enough to be responsible for postoperative hemorrhage in our experience, and the decline in white cell count returns, in the course of several hours, to a normal level.

Might I comment on some of yesterday's papers? I was extraordinarily interested in, and pleased to see, all the interest shown in the Forum program yesterday, and I regret that I left before the conclusion of the program as I assumed there would be no discussion. I merely wish to make one or two comments. I think that Melrose's suggestion of arresting the heart by injecting a potassium salt into the aorta between an aortic clamp and the aortic valves, should be an extraordinarily helpful one. The demonstration that all that is necesssary is to remove the clamp and wash out the coronary vessels in order to restore a normal beat, to my mind is very exciting and should prove very useful in intracardiac surgery of any sort, but particularly with by-pass through an extracorporeal circuit. Cardiac arrest should decrease the amount of cardiac venous blood which returns to the cardiac chambers, which will be helpful from a technical standpoint.

I was very much interested in Dr. Clowes's presentation. Kolff, ten years ago in Holland, told me that he was sure the artificial kidney would serve just as well as an artificial lung. I told him I rather doubted it. It has turned out that it is an extraordinarily difficult problem, as Dr. Clowes showed yesterday, and he is to be congratulated for his persistence in working with it. It is hard enough to get sufficient gas exchange between the fluid blood and the atmosphere with a free blood-gas interface, and if you interpose anything between the blood and the gas it is going to slow down the process considerably. Nevertheless, as Dr. Blalock said, ten years from now we are all going to look back upon our present devices as though they were old T-model Fords. The future of the artificial lung, which is the important part of the extracorporeal circuit (the pumping is easy), may well be what George Clowes has shown. If one can avoid a free blood-gas interface and still accomplish an adequate gas exchange, then an enormous step in advance will have been taken and we will have got rid of a lot of problems. I hope very much that he and others will stay with this problem and finally come up with something that resembles the pulmonary capillaries, difficult as that may be.

DR. F. D. DODRILL (Closing).—I wish to thank Dr. Gibbon for his discussion. The plasma hemoglobin on these patients with the total heart-lung by-pass, using the large volume flows, has varied from 100 to 150 mg. per cent for thirty minutes' duration. With the exchange transfusion at the conclusion of the procedure, we remove about 50 per cent of the increased plasma hemoglobin. In addition, we are removing approximately the same amount of deleterious substances, whatever they may be.

It is obvious that the blood is one of the most complex substances with which the surgeon deals. Twenty years ago a textbook of physiology might devote about two pages to the blood-clotting mechanism where as now as many as fifty pages might be devoted to this subject. Inasmuch as this whole field of blood physiology is rapidly expanding, this simple matter of diluting or removing a portion of these deleterious elements will, I hope, be of help to others during similar types of work.

# THE TREATMENT OF MITRAL INSUFFICIENCY BY THE PURSE STRING TECHNIQUE

## Initial Clinical Application

Robert P. Glover, M.D., and Julio C. Davila, M.D. (by invitation)
Philadelphia, Pa.

THE very considerable success in the surgical treatment of stenotic valvular disease of the heart in recent years has emphasized the urgency for the development of equally satisfactory methods for the correction of valvular insufficiencies. The mitral valve offers untold opportunity for the study and correction of its incompetent state just as this same valve did eight years ago when stenosis was first successfully relieved by surgical means.

Although a systolic murmur at the apex of the heart in the mitral valve area has long been recognized as perhaps the commonest of all valvular findings, many have considered for years, and have so stated, that mitral insufficiency is a relatively benign lesion and as such is rarely the cause of untoward clinical symptoms leading to death. The authors initially were prepared to accept this viewpoint during the early years of our surgical experience with mitral commissurotomy. Shortly, however, it became apparent all too frequently that the coexistence of mitral stenosis and insufficiency presented a clinical picture and a surgical problem which was quite different from that posed by the pure tight stenotic valve. This impression was strengthened when a review of our first consecutive 500 mitral commissurotomy cases revealed that the commonest incidence of either operative or late postoperative death occurred in those patients who either had pre-existing associated insufficiency unrelieved or worsened by commissurotomy or those in whom a pure state of stenosis was surgically transformed into a case of significant mitral insufficiency. It became even more obvious when, as time passed, a number of mitral valves were explored under the impression that stenosis was the predominant lesion only to find that very little if any stenosis was present and that indeed the entire syndrome of deterioration had been secondary to a relatively pure state of mitral insufficiency.

At the outset it was recognized that insufficiency of the mitral valve was undoubtedly a more insidious lesion than was stenosis but that in time it created even more serious effects to the myocardium of the left heart (left

---

From the Department of Thoracic and Cardiac Surgery at Presbyterian and Episcopal Hospitals and The Cardiovascular Research Laboratory at the Presbyterian Hospital, Philadelphia, Pa.

Research aspects of this program supported by U. S. Public Health Service Grant No. H-1712 (R).

Read at the Thirty-sixth Annual Meeting of The American Association for Thoracic Surgery at Miami Beach, Fla., May 7 to 9, 1956.

ventricle and atrium) than did its stenotic counterpart. Reports in the literature, together with some of our own more painful clinical experiences, indicated that the acute production of mitral insufficiency of significant degree was attended by almost immediate disaster both to the experimental animal and to the human patient. As an adequate experimental and/or clinical study had never satisfactorily been made and reported, concerning the pathophysiologic changes in chronic mitral insufficiency, this aspect of the over-all problem remained obscure except by inference and impression. Therefore, in 1952, on the thoracic surgical services at Episcopal and Presbyterian Hospitals and in the Cardiovascular Research Laboratory at Presbyterian Hospital, a concentrated program for the study of mitral insufficiency was inaugurated and has been carried out continuously to the present time.

## PATHOPHYSIOLOGIC CONSIDERATIONS

It goes without saying that before any concentrated study of the pathophysiologic derangements of an organ or, as in this case, a heart valve can be undertaken, a complete and thorough understanding of the normal anatomy and physiology must be basic knowledge. The first year of this program therefore was spent studying the normal anatomy of the mitral valve and its leaflets, the mitral annulus, the myocardial configuration at the atrioventricular juncture and the relationships of all other structures adjacent to the mitral valve and its annulus.[1] As many of the hearts examined were the seat of mitral valve disease, both stenosis and insufficiency, certain concepts as to the pathology of mitral insufficiency were gained together with a knowledge of its intricate anatomy.

With but minor variation it was learned that three basic features were present in the regurgitant mitral valve. *First,* and foremost, rheumatic valvular disease in such cases results in an absolute loss of valve tissue. As the aortic (septal) and the mural leaflets of the mitral valve contract within their substance they lose contact with each other and are no longer able to coapt during ventricular systole. As a rule this contraction is greatest in the mural leaflet which frequently becomes almost nonexistent. *Second,* and concomitantly, the chordae tendineae attached to the valve cusps become contracted, shortened, hypertrophied, and agglutinated tending further to fix the valve leaflets in a state of partial immobility. This effect increases the inability of the valve leaflets to coapt but it must be emphasized that even though contracted the leaflets (particularly the aortic) usually retained considerable pliability. Thus, during ventricular systole the mitral orifice remains open at a time when closure is necessary to properly and effectively direct the left ventricular outflow into the aorta. These two pathologic findings can obviously be termed *"an absolute loss of valvular occluding elements."* A *third* pathologic entity is almost routinely present. In a sense it might be considered a result of the first two although actually it too may appear concomitantly with the first two pathologic findings. As a result of the fact that the mitral valve leaflets cannot adequately close with each ventricular systole, a considerable vascular stream is propelled into the left atrium. For a considerable time it would seem, the left atrial chamber and its myocardium absorbs the effect of this jetlike shock by gradual dilatation and hypertrophy in so far as its myocardial elasticity will allow. The systolic jet and backpressure being intermittent and not sustained throughout the cardiac cycle (as it is in stenosis), the left atrium itself absorbs the total effect for an indefinite period and thus protects the pulmonary vascular bed and right heart from an increased pressure. In consequence, however, the left atrium enlarges constantly eventually to assume tremendous aneurysmal saclike proportions. Simultaneously the left ventricle, laboring to maintain an effective systemic output under impaired hydraulic conditions, is overworked. It responds by gradual dilatation and hypertrophy. Thus, as ventricle and atrium enlarge, the common point of their continuity—the mitral annulus or atrioventricular ring—does likewise, thus dilating the circumferential base to which the valve leaflets are attached and hinge. Thus, the *third* factor—dilatation of the mitral ring—is added to the production of mitral insufficiency. Now, not only are the contracted valve leaflets unable to close the mitral orifice but, with annular dilatation, the orifice to be closed becomes larger and a vicious cycle has been set up which cannot terminate other than by complete myocardial exhaustion. The concept, therefore, is not merely that the left heart myocardium wears out from mitral insufficiency of stationary amount and degree but rather is faced with an ever increasing load due to slow, gradual but constant enlargement of the mitral ring and orifice. Two factors are therefore at play—myocardial overwork and an ever increasing load (Fig. 1).

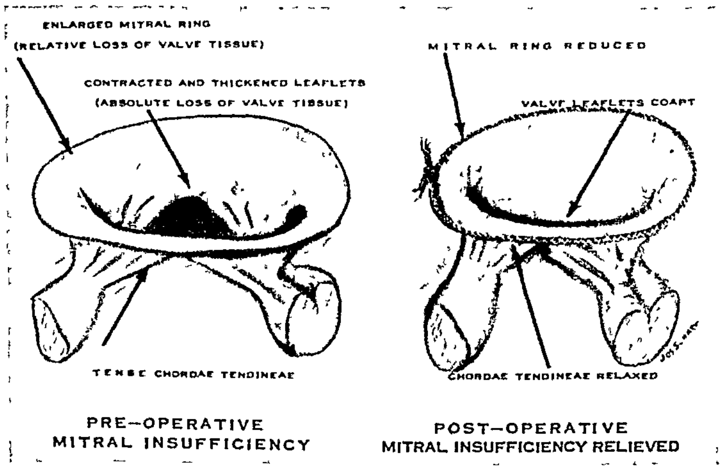

ENLARGED MITRAL RING
(RELATIVE LOSS OF VALVE TISSUE)
CONTRACTED AND THICKENED LEAFLETS
(ABSOLUTE LOSS OF VALVE TISSUE)
TENSE CHORDAE TENDINEAE
PRE-OPERATIVE
MITRAL INSUFFICIENCY
MITRAL RING REDUCED
VALVE LEAFLETS COAPT
CHORDAE TENDINEAE RELAXED
POST-OPERATIVE
MITRAL INSUFFICIENCY RELIEVED

Fig 1.—This diagram indicates the three major factors of pathologic deformity which contribute to lrawing on the left shows the valve and ring prior to circum' drawing on the right indicates the effect of the purse-string of annular size, relaxation of chordae tendineae and valve more complete coaptation of leaflet edges

With this concept in mind it seemed obvious that any surgical correction must be directed toward the relief of all three of these factors which play a part in the production of mitral valve insufficiency. Heretofore, and for that matter even to the present time, most of the methods advocated for the correction of mitral insufficiency took into account only the fact that there was loss of valve tissue. Attempts were, therefore, made to repair or replace or add to the missing valvular occluding elements. Techniques employing prosthetic devices,[2-8] grafts of pericardium or veins,[9-16] or methods of suturing the leaflets to each other were tried by several surgeons.[2, 9, 17] These procedures have been applied clinically to large numbers of unsuspecting patients despite the fact that little or no preliminary experimental work or evaluation was carried out. Further, in published reports, there is no objective evidence that these presumed corrective measures were effective. The clinical results have suggested, however, that such methods were unsatisfactory and disappointing and the eventual result in many of the patients has not been reported.

To the authors there seemed to be but one approach. As little could be done directly to the valve leaflets or their chordae tendineae, the one remaining approach was to modify the mitral annulus.[1, 18-20] A possible solution seemed to be based on the principle of reduction of the size of the orifice which the valve must occlude. This obviously could only be accomplished by controlled constriction of the atrioventricular ring or annulus (see Fig. 1). The concept was doubly attractive because it implied the avoidance of intracardiac foreign bodies and utilized to the full extent all available functional valvular occluding elements which remained, however scarred or puckered they might be. Further, if one accepts the observation that mitral insufficiency progressively increases as the annulus continues to dilate secondary to enlargement of the left heart chambers, circumferential annular constriction with its fixation of the mitral ring should abruptly terminate the increasing degree of regurgitation even though it may not be possible to totally abolish all of the original insufficiency. This factor is of vast importance, for no operation, however cleverly conceived, can be expected to completely remove in a routine manner all trace of insufficiency in the markedly diseased valves which one encounters in such cases. Any operation, therefore, which merely attacks a localized segment of the mitral annulus cannot be expected to overcome indefinitely the problem of future annular dilatation.

Two years were then spent in the animal research laboratory exploring the methods whereby an effective and at the same time innocuous circumferential suture could be placed about the mitral annulus. As might be expected, innumerable problems arose. The experiences encountered and the technical solutions employed for the final development of the operation now called "mitral purse-string" have been reported in previous communications. These data have clearly shown that experimental mitral insufficiency produced by almost every conceivable method can be routinely corrected by total annular constriction and documentary objective proof of this contention has been recorded. In addition to its ability to overcome the majority, if not all, of the mitral insufficiency present, it has been shown that mitral purse-string does not produce mitral stenosis, does not injure the effective myocardium, does not injure the coronary arterial system, and when properly placed does not cut through into the lumen of the left atrium or produce appreciable conduction defects. Animals, in whom mitral insufficiency has been produced experimentally and corrected by total controlled circumferential constriction of the annulus, have presently been followed for well over two years and some 20 such animals are being followed up to five years to determine the eventual status of a suture so placed.

### TECHNIQUE OF MITRAL PURSE STRING

The technique in its final form presently being employed in clinical mitral insufficiency, is outlined as follows. The patient is placed in the right lateral decubitus position with the left side up. The usual left posterolateral incision is made, entering the pleura through the bed of the resected sixth rib. Ribs five and seven are divided posteriorly if additional exposure is needed. This is often the case when one is dealing with the massively enlarged heart. The inferior pulmonary ligament is divided to the level of the inferior pulmonary vein ensuring adequate exposure posterior to the left ventricle. The pericardium is divided vertically, well posterior and parallel to the phrenic nerve, to expose the entire left heart from the level of the aortic arch to the diaphragm. In the far advanced case (and even in more minor cases in which there is considerable mitral insufficiency) the left atrium will be tremendous in size, often aneurysmal extending inferiorly to the diaphragm and well over to and even beyond the right border of the cardiac silhouette. A massive systolic thrill can be felt everywhere over the greatly dilated left atrial myocardium and this thrill can be traced out over the pulmonary veins into the pulmonary parenchyma. Indeed, on occasions the enlarged and hypertrophied left ventricle seems almost insignificant in size as it protrudes from this tremendous atrial mass. The heart may give the appearance of a large acorn with the left ventricle being the body of the nut and the left atrium being the caplike stem. In relatively pure mitral insufficiency, no other cardiac thrills will be felt. In associated mitral stenosis of significant degree a short diastolic thrill will be felt over the left ventricle near its apex. Routinely, the ascending aorta and the root of the aorta at the aortic valve ring should be palpated and compressed for evidences of aortic valve disease for ofttimes patients with a marked degree of mitral insufficiency will present clinically some evidence of a systolic murmur at the base often leading to the misdiagnosis of associated aortic stenosis. This is perhaps occasioned because the massive left atrium is in contact with the ascending aorta and transmits its systolic impulse or, as may also happen, the regurgitant stream through the open mitral valve is directed toward the interatrial septum and the jet impulse is transmitted upward to the aorta in this manner. Intracardiac pressure recordings are then taken in the left ventricle, left atrium, pulmonary artery, and aorta. It is extremely important that such pressure tracings be obtained for only in this way can one be objectively sure during surgery that cardiac hemodynamics have been affected by the operative procedure.

When the presence of significant mitral insufficiency has been ascertained in this manner the purse-string suture to be used is then prepared. A rectangular piece of pericardium is resected and sutured like a sleeve over the midportion of a long, double strand of standard, narrow cotton or umbilical tape. This sleeved portion need be no longer than 2 inches for it covers only that portion of the circumferential suture which lies in the transverse sinus from the point of emergency of the suture from the right atrium to the level of the circumflex coronary artery. Our experimental work suggests that it is important to bolster this area for should there be any tendency for the suture to cut through the annulus it will always be in this area. The use of the sleeve as described has prevented any such occurrence both experimentally and clinically.

As the suture is eventually applied immediately to the ventricular side of the great cardiac vein and circumflex coronary artery, the circumflex coronary artery just distal to the origin of its septal branch is partially isolated by tunnelling under the vessel so that the suture can eventually be drawn through (Fig. 2). The left atrial appendage is then purse-stringed in the usual manner, clamped and its tip opened. The right index finger is introduced into the left atrium and thorough exploration carried out. The usual finding in predominant mitral insufficiency, despite considerable leaflet contraction, scarring and thickening, is an aortic or septal leaflet of reasonable size and pliability unable to contact its opposing mural leaflet both by virture of its small size and the fact that it is held in a partially open state by contracted and foreshortened chordae tendineae. The posterior or mural leaflet, as a rule, is more badly diseased, considerably more contracted, and frequently presents merely as a thickened ridge emerging from the myocardium itself. Often there will be very minor agglutination of the two leaflets at the extreme angles but unless one or the other of the commissures is very well developed in the typical stenotic fashion, commissurotomy is not necessary or advised. The orifice of the mitral valve under these conditions will usually admit two to three fingers side by side and the mitral annulus can be recognized as an outer ring four to five fingerbreadths in diameter. Regurgitation of blood with each ventricular systole can be easily appreciated not as a swift, narrow jet, as is found in predominant stenosis, but more as a broad tongue or sheet of swiftly moving fluid. When insufficiency of this magnitude is present, it is impossible to estimate the amount of regurgitation present merely by finger palpation and appreciation. In predominant mitral insufficiency with little stenosis, the valve leaflets will usually have a considerable degree of pliability and very little if any calcium. With more major degrees of concomitant stenosis, one then finds more thickening, immobility, and calcific deposition. This is in line with the theory long held and often expressed by the author that calcium deposition is greatest in those valves which are immobile and are subjected to the incessant trauma of vascular flow pressure impinging upon leaflets partially fixed at right angles to the flowing stream of blood such as is seen in the valve deformity of stenosis.

Having ascertained the conditions present, the actual passage of the purse-string is then effected. The right index finger is removed and replaced by the left index finger. This finger is extended diagonally across the lumen of the left atrium and its tip comes to rest just on the atrial side of the crotch formed by the posteromedial angle of the valve orifice (Fig. 3). At this point the most medial aspect of the annulus can be appreciated. An assistant then gently elevates and rotates the left ventricle to a very minor degree *slightly* to the right. The diaphragm and the posterior leaflet of the pericardium are depressed. The operator can then clearly see the posterior aspect of the left atrium and left ventricle and can readily visualize the course of the posterior descending coronary artery and vein and the angle which these vessels form with the great cardiac vein and coronary sinus. The tip of the Davila* needle is then applied to the myocardium just below and to the left of this angle and advanced beneath the vessels and overlying fat which is invariably present. The intracardiac finger can immediately feel the pressure application of this needle and the further advancement of the needle is guided entirely by the intracardiac finger. The needle is passed in a tangential manner to the finger tip and myocardium and directed posteromedially and superiorly into the right atrium (see Fig. 3). This distance is as a rule no greater than 1 to 1.5 cm. or possibly 2 cm. in the largest of left atria. The needle or ligature carrier is entirely outside of the left atrial myocardium but pierces the wall of the right atrium as described. The assistant now allows the heart to return to its normal position, this period of very minor dislocation being never more than but a few seconds. The operator's left middle finger is then inserted into the depths of the transverse sinus where it comes to rest in the angle formed by the divergent walls of the two atria at the point where they combine to form the interatrial septum. The tip of the needle lying in the right atrium is directed to hug the interatrial septum and is guided onto the tip of the middle finger. At this point it lies between the intracardiac index finger tip and the tip of the middle finger. It is then gently thrust into the depths of the transverse sinus through the wall of the right atrium right at its juncture with the wall of the left atrium and interatrial septum. The needle is stabilized in this position and moved no further so that no kinking, displacement, or compression of the heart occurs. The wire which runs through the entire length of the Davila needle is then advanced and, as it does so, the eye of the needle is carried up from the depths of the transverse sinus out into the operative field where it can easily be threaded (Fig. 4). This feature of the needle prevents displacement or trauma to the heart during the act of threading. The end of the previously prepared purse-string is loosely tied to the eye of the needle, the wire withdrawn back into the depths of the transverse sinus and fixed again to the curved needle. The entire needle is withdrawn in parabolic reverse carrying with it the purse-string. The purse-string suture is pulled out from the posterior aspect of the heart until the sleeved portion of the purse-string suture has been snugged against the depths of the transverse sinus, very effectively tamponading the opening made in the right atrium by

---

*Available at The George P. Pilling and Son Company, Philadelphia, Pa.

Fig. 2.

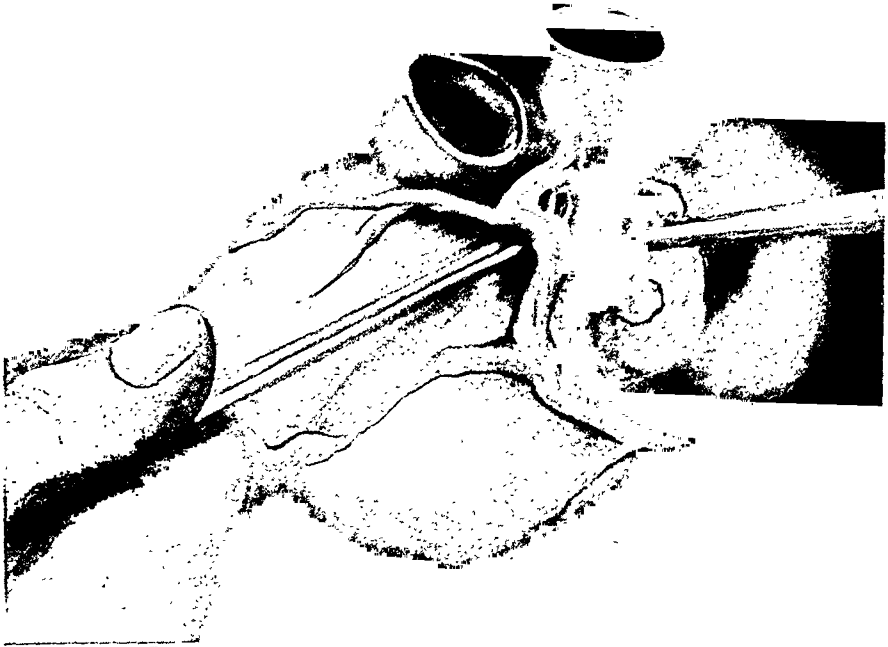
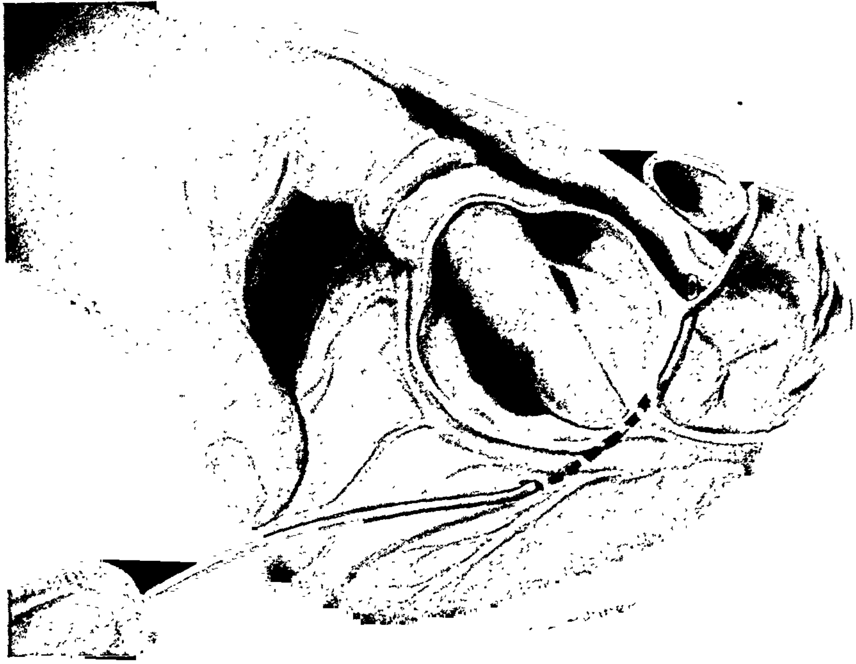


Fig. 3.



Figs. 2 to 7.—The technical details of the procedure are illustrated. As can be seen from these diagrams, the greatest part of this operation is actually done under direct vision from the outside of the heart. The exception to this is the maneuver of placing the medial or septal portion of the "purse-string." This is the crucial step of the technique and its execution must be thoroughly mastered in dogs before any clinical attempts should be made. Once this step of the operation is learned the procedure is quite simple.

Fig. 2.—Tunneling under the proximal portion of the circumflex coronary artery. This is done at a point about one cm. beyond the take-off of the anterior descending coronary artery.

Fig. 3.—Placement of the septal portion of the suture. The intra-atrial left index finger, and the left middle finger placed in the transverse sinus guide the needle. It is essential that the operator be clearly oriented in a three-dimensional sense as to the pertinent anatomy.

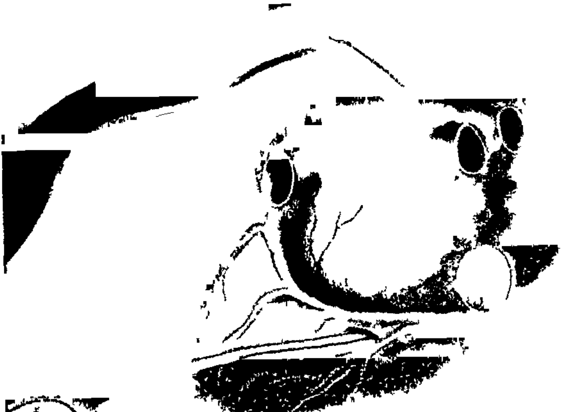


Fig 4

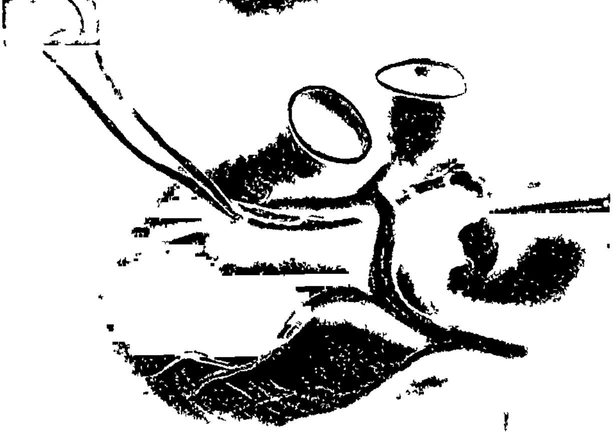


Fig 5

Fig 4 —After the tip of the needle has entered the transverse sinus under digital guidance the wire is then advanced so that the eye emerges anteriorly and can be easily threaded

Fig 5 —The pericardium covered portion of the suture is drawn into the transverse sinus and beneath the circumflex coronary artery in the previously constructed tunnel

Fig. 6.

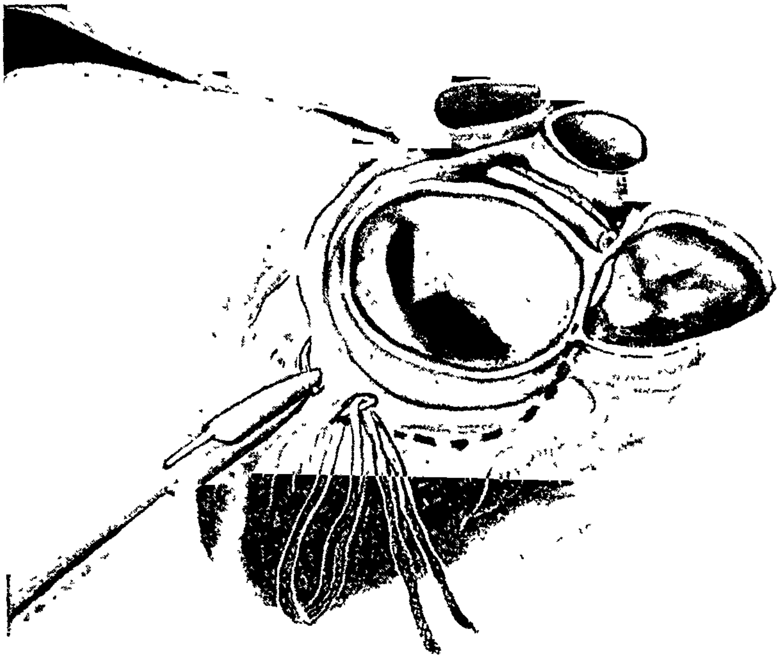


Fig. 7.

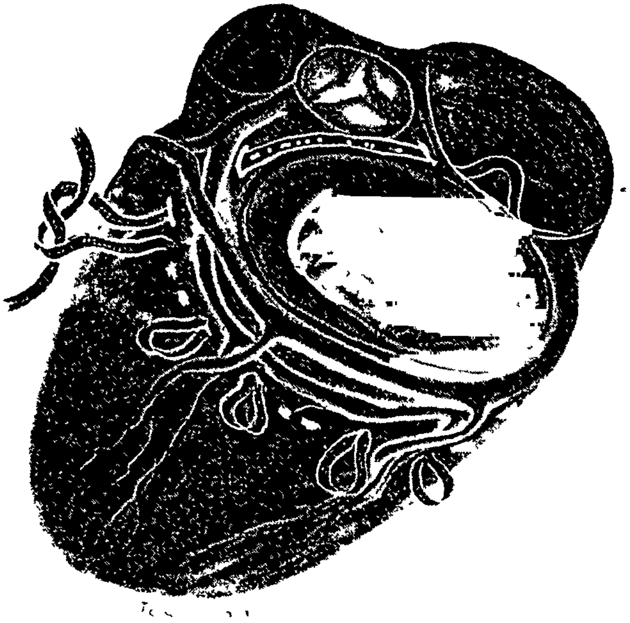


Fig. 6.—The posterior tail of the suture is then stitched into the atrioventricular fat pad beneath the circumflex coronary and cardiac vein and underpassing lateral descending branches of the circumflex. Coronary injury is easily avoided. The suture should lie within the fat pad and not in ventricular myocardium.

Fig. 7.—The suture completely placed and being tied anteriorly will lie concentrically over and just outside of the annulus. The suture is tied by the assistant as the operator palpates the regurgitant jet, the valve ring, and the valve leaflet action.

the passage of the suture (Fig. 5). Should there by any tendency to splitting of the thin myocardium in this area or should the emergence of the needle have been a little higher on the wall of the right atrium than desired, the tamponade and plugging effect of the sleeve of pericardium will effectively control any oozing. The anteromedial end of the circumferential suture is then drawn under the previously tunnelled circumflex coronary artery and the two ends now lie on the ventricular side of the mitral annulus. The posterior end of the suture is then brought forward to meet the anterior end by taking appropriate bites beneath the fat overlying the mitral annulus, taking care to avoid the left ventricular descending branches which arise from the circumflex coronary artery. Two or three such bites will accomplish approximation of the two ends of the circumferential suture (Fig. 6). At no point does the suture transfix myocardium except when entering and emerging from the right atrium.

The suture is then snugged in place by cinching down on the first throw of a square knot (Fig. 7). As this is done the suture eases into place in the V-like *groove formed by the interdigitating muscle fibers of the atrium and* ventricle and comes to rest exactly over the mitral annulus. The suture is further tightened, being guided in extent by the intra-atrial finger as it appreciates the diminishing degree of regurgitation resulting from coaptation of the valve leaflets. As a rule all regurgitation can be eliminated except a barely discernible whiff but this, of course, depends upon the pathologic status of the remaining valve tissues. The ring is usually reduced from a five-finger diameter to about a three-finger diameter and the effective valve orifice is not compromised. When maximum relief of regurgitation has been obtained, the square knot is completed and the mitral ring thereby fixed. The intra-atrial finger is then removed as the appendage is clamped. At this point, one frequently observes a diminution in the degree of left ventricular dilatation. What was formerly a large, distended, and incompressible left atrium is now frequently a flaccid, inelastic, easily compressible sac. Intracardiac pressures are repeated. The pressure in the left ventricle as a rule rises by 5 to 10 mm. Hg systolic and the pressures in the left atrium materially drop toward normal and not infrequently to within normal range. The original diffuse and intense systolic thrill felt everywhere over the left atrium is now either abolished or barely perceptible. The end diastolic pressure in the left atrium remains at a low level, indicating that no appreciable stenosis has been produced. In the average case, when the procedure has been properly performed, little, if any, cardiac irritability results other than an occasional ectopic ventricular beat. Frequently the cardiac rate, previously rapid, is now reduced to normal levels and is *visibly more steady and effectual.*

The appendage is ligated at its base, a portion of its tip amputated for histologic study, and the remainder oversewn. The operative field is flushed with warm saline, the pericardium partially closed and now, even though a portion of the pericardium has been resected, there is usually no difficulty in completely containing the heart loosely within the long, overstretched *pericardium. A small opening is left in the pericardium* to prevent

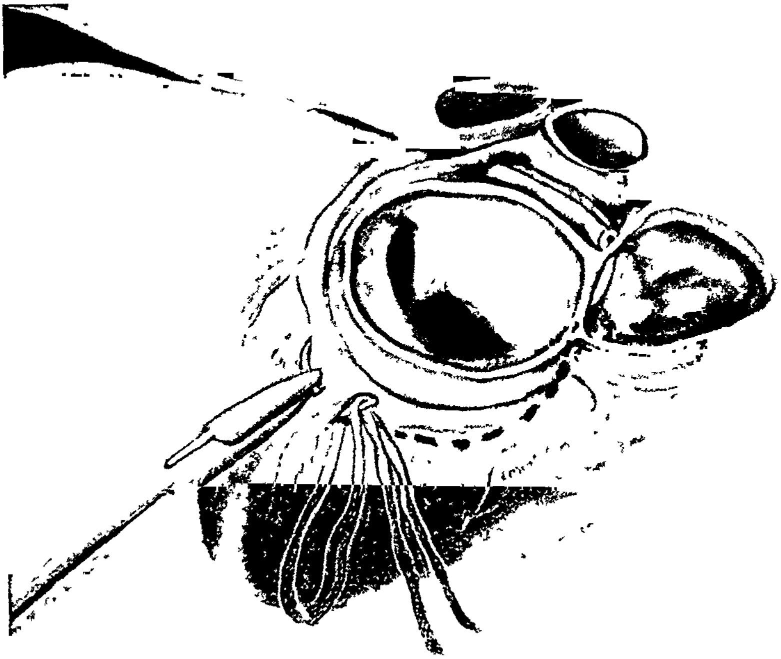


Fig. 6.

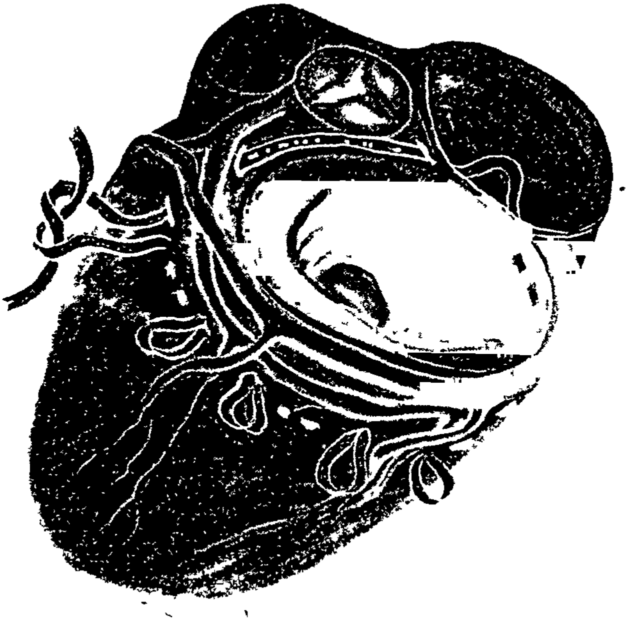


Fig. 7.

Fig. 6.—The posterior tail of the suture is then stitched into the atrioventricular fat pad beneath the circumflex coronary and cardiac vein and underpassing lateral descending branches of the circumflex. Coronary injury is easily avoided. The suture should lie within the fat pad and not in ventricular myocardium.

Fig. 7.—The suture completely placed and being tied anteriorly will lie concentrically over and just outside of the annulus The suture is tied by the assistant as the operator palpates the regurgitant jet, the valve ring, and the valve leaflet action

the passage of the suture (Fig. 5). Should there be any tendency to splitting of the thin myocardium in this area or should the emergence of the needle have been a little higher on the wall of the right atrium than desired, the tamponade and plugging effect of the sleeve of pericardium will effectively control any oozing. The anteromedial end of the circumferential suture is then drawn under the previously tunnelled circumflex coronary artery and the two ends now lie on the ventricular side of the mitral annulus. The posterior end of the suture is then brought forward to meet the anterior end by taking appropriate bites beneath the fat overlying the mitral annulus taking care to avoid the left ventricular descending branches which arise from the circumflex coronary artery. Two or three such bites will accomplish approximation of the two ends of the circumferential suture (Fig. 6). At no point does the suture transfix myocardium except when entering and emerging from the right atrium.

The suture is then snugged in place by cinching down on the first throw of a square knot (Fig. 7). As this is done the suture eases into place in the V-like groove formed by the interdigitating muscle fibers of the atrium and ventricle and comes to rest exactly over the mitral annulus. The suture is further tightened, being guided in extent by the intra-atrial finger as it appreciates the diminishing degree of regurgitation resulting from coaptation of the valve leaflets. As a rule all regurgitation can be eliminated except a barely discernible whiff but this of course depends upon the pathologic status of the remaining valve tissues. The ring is usually reduced from a five-finger diameter to about a three-finger diameter and the effective valve orifice is not compromised. When maximum relief of regurgitation has been obtained, the square knot is completed and the mitral ring thereby fixed. The intra-atrial finger is then removed as the appendage is clamped. At this point, one frequently observes a diminution in the degree of left ventricular dilatation. What was formerly a large, distended and incompressible left atrium is now frequently a flaccid inelastic, easily compressible sac. Intracardiac pressures are repeated. The pressure in the left ventricle as a rule rises by 5 to 10 mm. Hg systolic and the pressures in the left atrium materially drop toward normal and not infrequently to within normal range. The original diffuse and intense systolic thrill felt everywhere over the left atrium is now either abolished or barely perceptible. The end diastolic pressure in the left atrium remains at a low level, indicating that no appreciable stenosis has been produced. In the average case, when the procedure has been properly performed, little, if any, cardiac irritability results other than an occasional ectopic ventricular beat. Frequently the cardiac rate, previously rapid, is now reduced to normal levels and is visibly more steady and effectual.

The appendage is ligated at its base, a portion of its tip amputated for histologic study, and the remainder oversewn. The operative field is flushed with warm saline, the pericardium partially closed and now, even though a portion of the pericardium has been resected, there is usually no difficulty in completely containing the heart loosely within the long, overstretched pericardium. A small opening is left in the pericardium to prevent the entrapment of pericardial fluid. The lung is completely expanded, a lung biospy is obtained, the pleura drained by catheter, and the chest wall closed in the usual manner. The procedure will have been performed with little, if any, more trauma than is seen in the average mitral commissurotomy. Should appreciable stenosis have been present, an appropriate guillotine-type commissurotomy, either unilateral or bilateral, will be performed to free the leaflets from each other, thus ensuring maximum motion of valve tissues. The commissurotomy is best performed after the purse-string has been placed in its entirety but just before it is tied. As a rule, after commissurotomy, even though the intracardiac finger may now be unable to appreciate any increase in regurgitation, an interatrial pressure recording will almost routinely reveal that the pressure within the atrium has been very materially increased. This finding has proved to us over and over again that, in the presence of large amounts of regurgitation, intra-atrial finger estimation of the degree of regurgitation present is worthless. After commissurotomy the annular purse-string is tied down to its maximum degree as described. The entire procedure is monitored by a continuous oscillographic image and frequent recordings of the electrocardiogram.

## SELECTION OF PATIENTS

Having developed an effective technique for the performance of total circumferential constriction of the mitral annulus and observed its effect in over 250 animal experiments over a period of two years, it logically followed that clinical application was in order. Ideally, from a purely surgical salesmanship viewpoint, it would have been highly desirable to employ the procedure on those patients exhibiting more minor degrees of mitral valve incompetence. A low operative mortality and a high percentage of living patients might attest the fact that the operation could be at least tolerated (improvement or no improvement), but was there justification at this point to subject patients in reasonable clinical health to the hazards of any new and intricate procedure of this magnitude? Under these circumstances would maximum objective data be forthcoming to ensure an honest and accurate appraisal of the procedure? We think not. On the contrary, it was felt that such application should not be employed until the efficacy of the procedure was entirely proved on the badly damaged rheumatic heart for, not infrequently, those procedures which seem to be highly effective in the laboratory prove to be less desirable or even detrimental to the human disease complex. Therefore, it was decided that initial clinical application would be employed only in those patients showing far-advanced, even terminal, degrees of rheumatic heart disease with mitral insufficiency. Thus, a number of patients previously denied surgery because of the hopeless nature of their cardiovalvular disability (primarily due to insufficiency) were recalled, the facts and status of the operation presented and then subjected to surgery as desired. It was determined that this pilot study should not include more than 25 to 30 patients. It was fully realized that due to the desperate condition of these patients, perhaps few would eventually survive or be materially helped. Nevertheless the hope was ever present that some appreciable salvage might result.

### ANALYSIS OF PATIENTS

Twenty-seven patients with rheumatic mitral insufficiency, pure or in combination with mitral stenosis, have undergone "mitral purse-string" to date. In 6, a concomitant commissurotomy was likewise performed and in 3 a commissurotomy had been performed one to two years before. The first mitral purse-string in the series was carried out on Jan. 20, 1955, and this patient is living a normal life today, sixteen months later. Twenty-four were female and 3 were male; the youngest was aged 7 and the oldest aged 52. The average age was thirty years.

*Clinical Status.*—Each of the 27 patients was in an advanced stage of chronic congestive failure (Stage IV). Fourteen were classified as intractable (terminal) failure (Group I), for despite the most diligent and heroic of medical regimens, their congestive failure could not be abolished and these patients came to surgery with variable degrees of pulmonary congestion, hepatomegaly, often with ascites and evidences of peripheral edema and stasis. Admittedly, patients in such a state of myocardial degeneration have long been recognized as the poorest of candidates for surgery with little hope of salvage. The remaining 13 patients (Group II) were likewise in an advanced state of myocardial degeneration and chronic congestive failure but by dint of tedious and prolonged medical therapy they came to surgery clinically free

TABLE I. CARDIAC CATHETERIZATION DATA
(PREOPERATIVE)

| PATIENT | GROUP | RIGHT ATRIUM | RIGHT VENTRICLE | PULMONARY ARTERY | PULMONARY VENOUS CAPILLARY | ARTERIAL (BRACHIAL OR FEMORAL) |
|---|---|---|---|---|---|---|
| 1. R. M. | II | | | | | 125/75 |
| 2. B. F. | I | 25/14 | 83/10 | 80/47 | 20/11 | |
| 3. P. F. | II | | 30/0 | 25/5 | 14/11 | 125/65 |
| 4. B. W. | I | 15/7 | | 58/30 | | |
| 5. L. T. | I | 8.5/2 | 75/-1 | 88/35 | | |
| 6. R. S. | I | 1.5/-3 | 20/-5 | 28/10 | 16/5 | 125/62 |
| 7. J. L. | I | 1/-3 | 31/-6 | 30/11 | 16/-1 | 98/57 |
| 8. M. G. | I | 7/0 | | | | |
| 9. J. M. | I | 10/6 | 80/2 | 76/36 | 29/15 | |
| 10. H. S. | I | | 48/0 | 46/11 | 49/17 | |
| 11. A. T. | II | | | | | |
| 12. E. J. | II | 3/-2 | 23/-4 | 20/4 | | 130/60 |
| 13. G. S. | II | (9) | 50/10 | 55/20 | (16) | 118/75 |
| 14. P. B. | I | 5/-1 | 64/0 | | | |
| 15. K. K. | II | 3/-4 | 12/1 | 12/5 | 10/6 | |
| 16. R. R. | I | 4/-3 | 42/-3 | 45/18 | | |
| 17. F. C. | II | 5/0 | 35/-5 | 30/5 | 12/0 | 140/80 |
| 18. F. M. | II | 9/4 | 38/7 | 38/18 | | 118/69 |
| 19. G. B. | II | 9/2 | 34/5 | 28/11 | 26/9 | 120/58 |
| 20. J. K. | I | 10/6 | 35/5 | 35/17 | | 140/48 |
| 21. J. S. | I | 14/10 | 60/0 | 60/20 | 30/18 | 120/64 |
| 22. G. M. | II | -3/-10 | 35/-7 | 40/6 | 15/0 | 124/76 |
| 23. D. W. | I | | 60/0 | 60/18 | 40/12 | 120/80 |
| 24. E. H. | II | 13/5 | 92/5 | 92/44 | 52/22 | 140/84 |
| 25. H. M. | I | 24/15 | 84/10 | 80/40 | 52/22 | 126/80 |
| 26. M. L. | II | 7/2 | 40/3 | 36/13 | 32/12 | 120/70 |
| 27. G. L. | II | | 38/7 | 37/18 | 30/12 | 120/60 |

of decompensation. The contrasting results obtained in these two groups of patients have been extremely enlightening and have served to corroborate further the impressions gained as to the indications for surgery in valvular disease over the years in our rather extensive experience with the rheumatic heart.

*Preoperative Studies.*—Each patient was studied in the usual routine cardiac fashion familiar to all, including routine blood work, blood chemistry, urinalysis, electrocardiograms, teleoroentgenograms and fluoroscopy, and phonocardiograms. In 25 of the patients a satisfactory cardiac catheterization was accomplished and these data have been summarized in Table I. It will be noted that in one half of the patients catheterized the pulmonary vascular and right heart pressures were two to four times higher than normal. Hypertension in the lesser circuit of this degree, while a common finding in mitral stenosis, is distinctly uncommon in the average case of mitral insufficiency

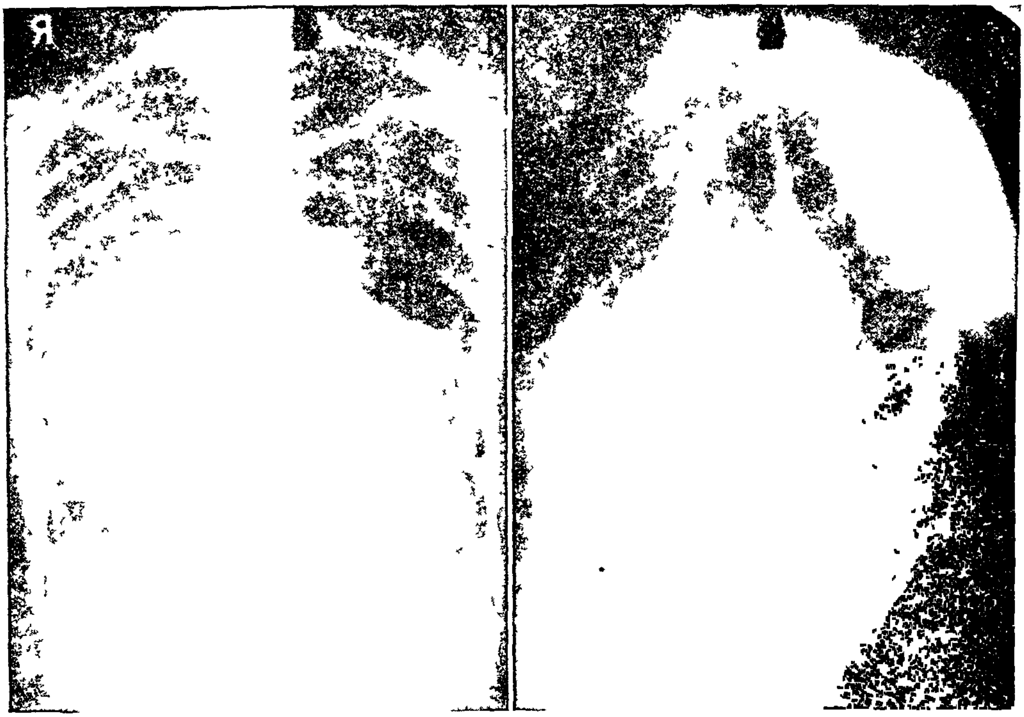


Fig 8.—Posteroanterior and left lateral teleoroentgenograms of Case 5 which typify the findings in cases of Group I

The degree of cardiac enlargement shown radiographically in each patient was marked. A typical example of this type of cardiac enlargement characteristic of the patient with mitral insufficiency in Group I of this series is shown in Fig 8

The changes in the electrocardiogram of each patient were extensive and most variable This aspect of the findings in these patients has formed the subject of another communication to be presented shortly. It is sufficient to state here that, in 25 patients, atrial fibrillation was present and in the remainder a normal sinus rhythm persisted. In 3 patients an atrioventricular block was noted. Variable degrees of atrial hypertrophy and combined ventricular hypertrophy were routinely present.

Pre- and postoperative phonocardiograms routinely obtained confirmed the clinically obvious diminution in the mitral systolic murmur as shown in Fig. 11.

*Group I—Intractable (Terminal) Failure.*—There were 14 patients in this category. Five of these patients had undergone previous mitral valve exploration one to two years before, and in 2 of these a commissurotomy had been performed. Needless to say, in each of these 5 patients the performance of a "mitral purse string" was doubly difficult for all intrapericardial landmarks were obliterated as were the left atrial appendages. The postoperative changes encountered were directly responsible for the operative death of one of these for an uncontrolled hemorrhage was produced while attempting to open the adhesion-obliterated transverse sinus.

Seven of the 14 patients died within the first month and as such must be considered as operative deaths. In 2, ventricular fibrillation developed as the pericardium was being opened and these hearts could not be resuscitated. Two others went into ventricular fibrillation during the initial maneuvers of the passage of the purse string. Cardiac massage and electric shock were unsuccessful in these until the purse string was completely passed and tied rendering the mitral valve competent. Massage at this point effectively restored cardiac output and coronary filling so that defibrillation resulted. Despite this the patients were lost. The fifth patient tolerated the operative procedure surprisingly well despite her precarious condition only to succumb one week later to progressive irreversible heart failure. The remaining 2 died of operative hemorrhage, one for reasons alluded to above and the other as a result of a tear in the right atrium when the Davila needle emerged into the transverse sinus too high on the wall of the right atrium. These latter two deaths could well be classified as technical errors.

It must be apparent from the brief descriptions above that the majority of the deaths described were occasioned primarily by the far advanced nature of the patient's disease. One can hardly blame the operation of circumferential annular constriction for the immediate effect of this procedure, hemodynamically speaking, was accomplished in but one of the 7.

The remaining 7 patients survived surgical intervention although in 2 ventricular fibrillation was present for twenty-four and sixty-five minutes respectively. Both of these were restored to their preoperative rhythm only after the mitral valve had been made competent by circumferential constriction. One patient after rather considerable improvement in her congestive state during the first postoperative week shortly reverted to her previous intractable failure and died two weeks later. At post-mortem examination an extensive calcific aortic stenosis was noted. The presence of aortic stenosis had been recognized at the time of surgery but its degree of severity was misjudged so that this valve was not opened. Even had an aortic commissurotomy been considered, it is doubtful that the patient would have survived the added procedure. Two patients obtained rather remarkable relief from their

TABLE II. INTRACARDIAC PRESSURES BEFORE AND AFTER PURSE-STRING OBTAINED AT SURGERY

| | INITIAL | | | | | AFTER COMMISSUROTOMY | | | AFTER ATRIOVENTRICULAR PURSE-STRING | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| PATIENT | LEFT ATRIUM | LEFT VEN-TRICLE | AORTA | PULMO-NARY ARTERY | BLOOD PRESSURE | LEFT ATRIUM | LEFT VEN-TRICLE | BLOOD PRES-SURE | LEFT ATRIUM | LEFT VEN-TRICLE | AORTA | PULMO-NARY | BLOOD PRESSURE | CLINICAL GROUP |
| †1. R. M. | 30/10 | 108/-10 | | | | | | | 19/6 | 106/-3 | | | | II |
| 2. B. F. | 78/15 | 110/10 | | 110/65 | 105/95 | | | | 17/7 | 115/6 | | 102/58 | 110/100 | I |
| †3. P. F. | 24/6 | 112/22 | | | | | | | 22/11 | 160/-5 | | | | II |
| 4. B. W. | 28/11 | 155/-2 | 130/65 | 54/25 | 130/15 | | | | 18/6 | 150/-2 | 105/62 | 54/26 | 120/110 | I |
| 5. L. T. | 38/5 | 102/-5 | 110/80 | 110/70 | 110/80 | | | | * | | | | | I |
| 6. R. S. | 27/7 | 80/-8 | | 48/12 | 120/70 | | | | 10/0.5 | 96/-4 | | | 110/70 | I |
| 7. J. L. | 20/-3 | 90/-8 | | 25/12 | 110/90 | | | | 5/-4 | 92/-10 | | 24/7 | 110/80 | I |
| 8. M. G. | 48/13 | 128/0 | | 120/60 | 120/80 | | | | 25/7 | 112/0 | | 94/44 | 140/80 | I |
| 9. J. M. | 100/60 | 128/70 | | 76/3 | 105/60 | | | | * | | | | | I |
| 10. H. S. | 34/14 | 115/0 | | 33/10 | 90/80 | | | | * | | | | | I |
| 11. A. T. | 47/-10 | 100/-8 | | 30/0 | 90/70 | | | | 0/-10 | 100/-10 | | 18/0 | 80/70 | II |
| 12. E. J. | 48/8 | 160/0 | | 48/16 | 180/20 | | | | 15/10 | 140/0 | | | | II |
| †13. G. S. | 29/7 | 138/-8 | 128/78 | 116/60 | 130/100 | | | | 8/1 | 138/-2 | 132/64 | | 130/100 | II |
| 14. P. B. | 44/10 | 85/0 | 88/50 | 52/24 | 110/80 | 58/10 | | | 27/8 | 90/4 | 90/56 | 38/12 | 100/80 | I |
| †15. K. K. | 45/5 | 116/4 | | 35/15 | 110/80 | | | | 20/10 | 120/0 | | 35/21 | | II |
| 16. R. R. | * | | | | | | | | | | | | | I |
| †17. F. C. | 30/2 | 110/-10 | | 25/4 | 130/100 | | | | 14/1 | 110/0 | | 24/4 | 125/90 | II |
| †18. F. M. | 48/7 | 120/0 | | | 125/105 | | | | 20/10 | 120/0 | | | | II |
| †19. G. B. | 30/12 | 140/8 | 100/80 | | 120/80 | | | | 20/10 | 100/0 | | | 110/90 | II |
| 20. J. K. | 35/10 | 110/-4 | 125/75 | | | 70/? | 84/-8 | | * | | | | | I |
| 21. J. S. | 55/8 | 78/-4 | | | | | | | * | | | | | I |
| †22. G. M. | 35/5 | 100/-4 | | | 100/80 | | | | 25/15 | 120/5 | | | 130/90 | II |
| †23. D. W. | 50/7 | 125/7 | | | 125/? | | | | 27/7 | 130/7 | | | 130/? | I |
| 24. E. H. | 60/18 | 120/7 | 124/84 | | 115/70 | 92/18 | 100/18 | | 40/20 | 120/18 | 120/84 | | 110/70 | II |
| 25. H. M. | 64/12 | | | | | | | | 64/25 | | | | | I |
| †26. M. L. | 30/12 | 104/3 | 112/68 | | 100/70 | | | | 25/12 | 100/5 | 104/68 | | 100/70 | II |
| †27. G. L. | 43/15 | 124/4 | 100/60 | | 100/70 | 52/13 | 100/6 | 70/60 | 33/15 | 120/4 | 120/64 | | 120/80 | II |

*Pressures not taken due to operative difficulties

†Living patients to date.

preoperative congestive states. Indeed, all signs of congestive failure disappeared and they were restored to a considerable degree of normal activity. Both of them died rather suddenly and in an entirely unexpected, unexplained manner, one at three months and one at five months. Both had been very active and one had just returned home from a shopping tour. Post-mortem examination in each failed to reveal any cause of death. The hearts of these patients were referred to us for examination and in each the purse-string suture was accurately and well placed with no evidence of either myocardial or coronary damage noted. Both valves had been restored to a high state of competency by the purse-string procedure. Three other patients likewise improved very dramatically for a matter of weeks and months only to revert again to a state of slowly progressive congestive failure. These 3 patients eventually died at six weeks, two months, and eight months, respectively. Post-mortem examinations were performed in 2 of these and again it was noted that the purse-string suture was properly and accurately placed and had accomplished very effective improvement in valvular competency. It is of interest that the patient in this group who lived the longest was the one who survived sixty-five minutes of almost continuous ventricular fibrillation. Constant and apparently very adequate massage was maintained throughout for little if any cerebral damage resulted. The final patient is alive today, three months after surgery, and is considerably improved. In the light of her 13 predecessors as just described it is altogether probable that she too will eventually succumb to intractable failure for her original cardiac status was quite as bad as that of the others.

It goes without saying that the results from a clinical standpoint were most disheartening. No appreciable salvage was obtained other than the fact that the survivors were made more comfortable for most of their remaining days. On the other hand a great deal of highly valuable hemodynamic and technical information was obtained which has gone far toward the establishment of this procedure as an effective means of controlling mitral insufficiency for patients in an earlier stage of this condition. The following points have been clarified. (1) Once the technique has been mastered, it can be accomplished under the most harassing circumstances and under conditions of almost impossible extremes of pathologic complication. It is tolerated amazingly well even in the most precarious of patients. (2) Post-mortem data are available in 11 of the 13 patients who have died. In each of these examined hearts, the purse-string suture had been accurately placed and lay directly over the mitral annulus. By simple laboratory test and by observation of these hearts on our pulse duplicator[21] which allows for direct vision of valves in action, the mitral valve in all but 2 cases was corrected by at least 80 per cent. In one of the two uncorrectable valves there was essentially no valve tissue present. Both the aortic and mitral leaflets were so small from contraction and induration that no coaptation could be obtained short of producing an almost complete stenosis of the mitral annulus (Case 25). The other (Case 9) had undergone commissurotomy elsewhere one year previously. At that time the aortic leaflet of the mitral valve had been split in its exact center rather than an opening made at the presumed anterolateral commissure. The patient developed, as might be expected, a tremendous regurgitation and why he did not die immediately is a complete enigma for, when such is done experimentally, death is prompt. Nevertheless, this patient did survive only to become a complete invalid from constant congestive failure. The purse-string was passed without difficulty but because the continuity of the aortic leaflet had been destroyed, purse-stringing under these conditions could coapt the aortic leaflet to the mural leaflet but could not coapt the bisected halves of the aortic leaflet itself. This patient died from unrelievable mitral insufficiency.

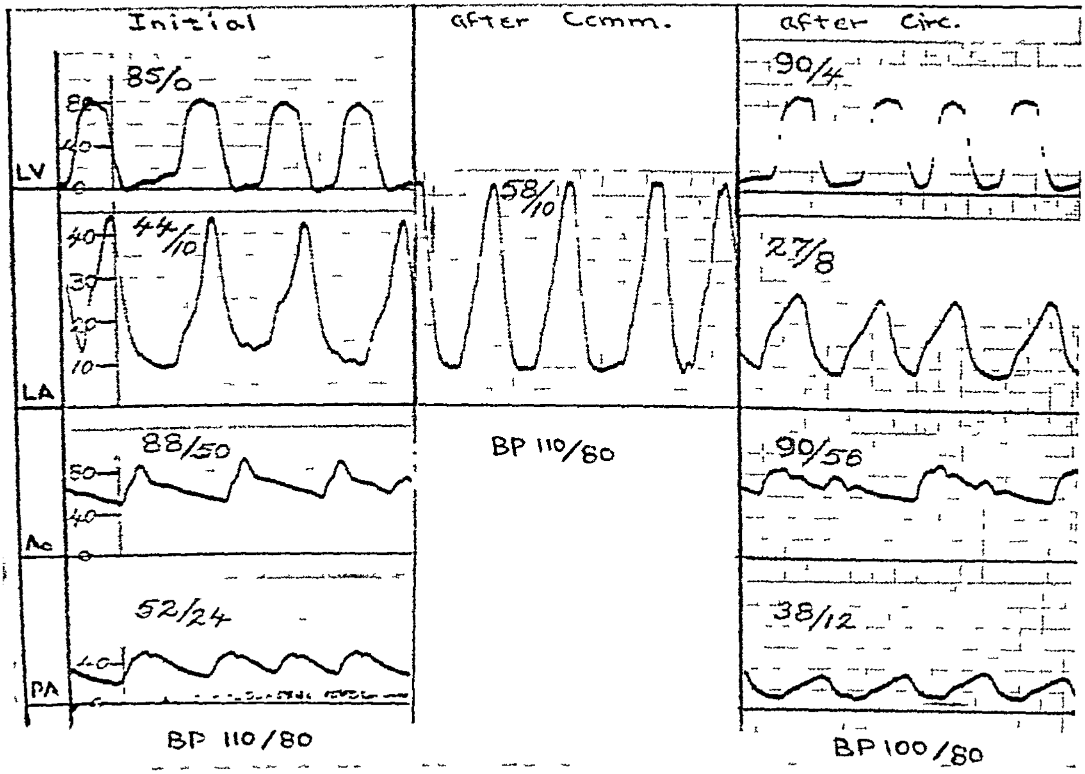


Fig 9.—Pressure pulse tracings taken at surgery in a patient with mitral insufficiency and mitral stenosis (Case 14). In the initial tracings note the low ventricular and aortic pressure, the typical regurgitant contour and pressure range of the atrial tracing, and the elevated pulmonary artery pressure. Note that after commissurotomy there is a marked increase in the left atrial pressure. The regurgitant jet palpated by the exploring finger is not a reliable indication of whether regurgitation has been aggravated or improved by commissurotomy. After placement and tieing of the "purse-string," the ventricular and aortic pressures rise, the left atrial pressure falls, and the contour changes favorably, the pulmonary artery pressure falls. In most instances the change noted in pulmonary artery pressure may be small but in all the cases studied the left ventricular, aortic, and atrial changes are consistent.

(3) Proof of the above statements is shown in Table II. The reduction in left atrial pressure was highly satisfactory in all but the above-mentioned cases (Cases 9 and 25). Pressure curves in the left atrium likewise were restored to a more normal contour as were those obtained from the left ventricle (Fig. 9). (4) In no instance were the coronary arteries compromised, no erosion into the left atrium occurred, there were no appreciable myocardial changes and no stenosis was produced.

These data were essential to the proper continuation of this program.

*Group II—Chronic Congestive Failure Clinically Correctable.*—There were 13 patients in this category. One of these patients had undergone previous mitral valve exploration two years before but insufficiency and not stenosis had been found. Re-exploration of this patient at this time did not present the technical difficulties noted in the 5 similar cases in Group I.

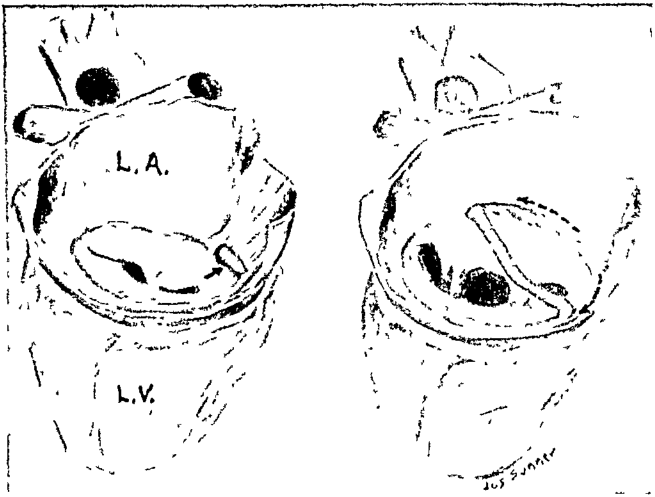


Fig. 10.—Drawing of the findings at autopsy in Case 11. The illustration on the left shows the position of the suture. Penetration into the lumen of the left atrium in this manner constitutes a serious technical error which can be avoided by careful palpation of the posterior commissural area of the ring after placement of the suture and withdrawal of it if it should be so placed. It is essential that in introducing the needle from the posterior aspect of the heart it be directed correctly into the right atrium. The figure on the right illustrates the erosion of the suture in two directions on the circumference of the ring beginning at the points of perforation (indicated by the dotted arrows). When the suture is properly placed, erosion does not occur.

Two of the 13 patients died within the first month and were considered operative deaths. One of these should have obtained a highly satisfactory result judging from the favorable nature of the valve leaflets and from the extremely satisfactory reduction in the left atrial pressure (Case 12). Inexcusably but inadvertently she received 500 c.c. of totally incompatible blood and passed away rather promptly after surgery from overwhelming intravascular hemolysis. The other patient was the second example in this series of a patient who, for all intents and purposes, had no remaining effective valve tissue. The aortic leaflet was but one centimeter in radius and most of the mural leaflet was nonexistent. At the posteromedial extremity of the mural leaflet there was a small amount of valve tissue which was attached to a short chordae tendineae that had long since been ruptured and this tiny portion of valve was flail-like. Even so, mitral purse string did accomplish some reduction of valvular insufficiency although left atrial pressures still remained at a much higher level than that seen in all the other cases in this group (Case 24). This patient died unexpectedly on the fourth postoperative day following a period of bradycardia. Post-mortem examination could establish no cause of death. One additional patient in this group died three months after surgery from subacute bacteral endocarditis. Post-mortem examination revealed that this patient had been the victim of the one truly technical error in the entire series. Fig. 10 illustrates that during the passage of the purse-string suture a tiny portion lay exposed within the lumen of the left atrium, at the posteromedial commissural area. As time passed this portion of the suture had cut well out into the lumen of the left atrium and thus lay like a strut across the lumen of the atrium. Extensive thrombosis had developed on this nidus leading to an uncontrollable blood stream infection. This case points up the extreme necessity for accurate placement of the purse-string for if a segment has been misplaced in the lumen of the left atrium, the result will be as just cited. This has been observed on a number of occasions during the early experimental animal work. In contrast to this, however, that portion of the suture which lies in the right atrium, because it is snugly against the interatrial septum and is not subject to the development of a cross luminal position, rapidly becomes endothelized and buried, a process which takes but two to three weeks. In all post-mortem specimens examined, this right atrial segment of the circumferential suture was buried in this fashion.

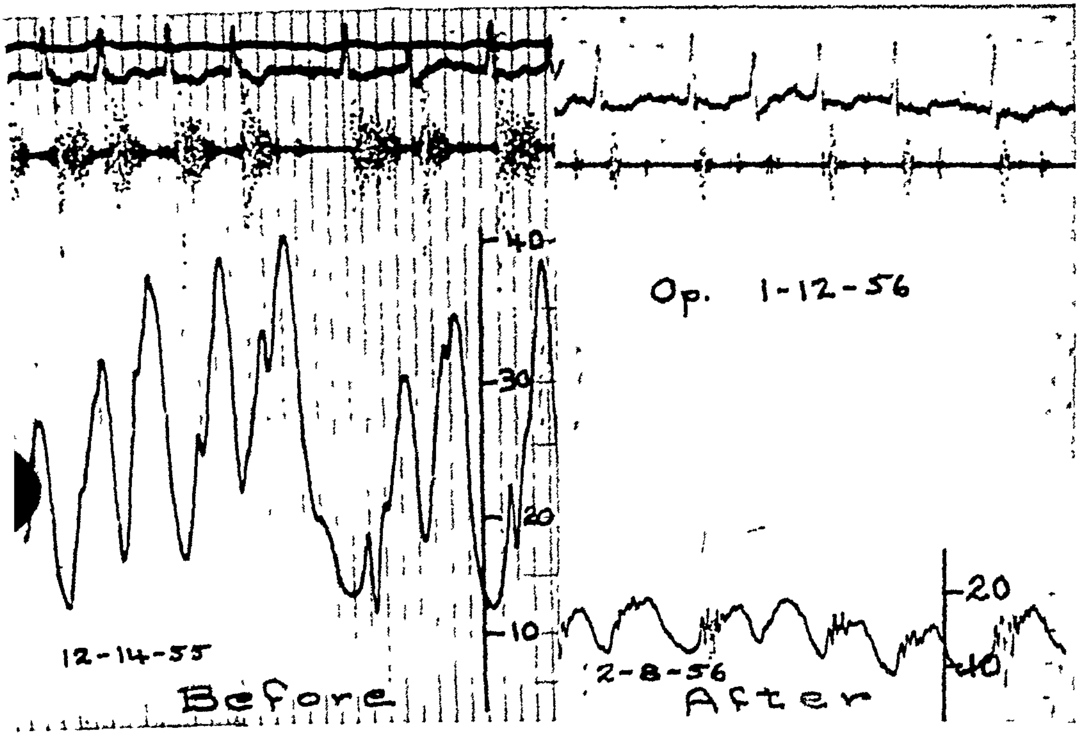


Fig. 11.—Left atrial pressure pulse r[illegible] ft atrial puncture approximately one month before and one m[illegible] of mitral insufficiency. Note the pre- and postoperative [illegible] the atrial tracing (Case 18). (Courtesy of Dr. A. G. Morrow, National Heart Institute, Bethesda, Md.)

The remaining 10 patients are living to date. Two of these are sixteen months postoperative (Cases 1 and 3), 2 at eight months (Cases 13 and 15), one at six months (Case 17), 2 at five months (Cases 18 and 19), one at four months (Case 22), and 2 at two months (Cases 26 and 27). Those 5 patients who are now living six months to one and one half years after surgery have obtained rather excellent results, are living relatively normal lives and are carrying out a full schedule of household activity. Much the same can be said for those under six months but not enough time has elapsed to make a definite statement to that effect.

Objective evidence of the correction of mitral insufficiency and the subsequent improvement of patients so treated is available in the form of pre- and postoperative phonocardiograms, teleroentgenograms, and left atrial pressures taken before and after mitral purse string at the time of surgery. As yet, none of these patients has been subjected to a formal postoperative cardiac catheterization.

An example of the type of pre- and postoperative phonocardiographic tracing routinely obtained is shown in Fig. 11.

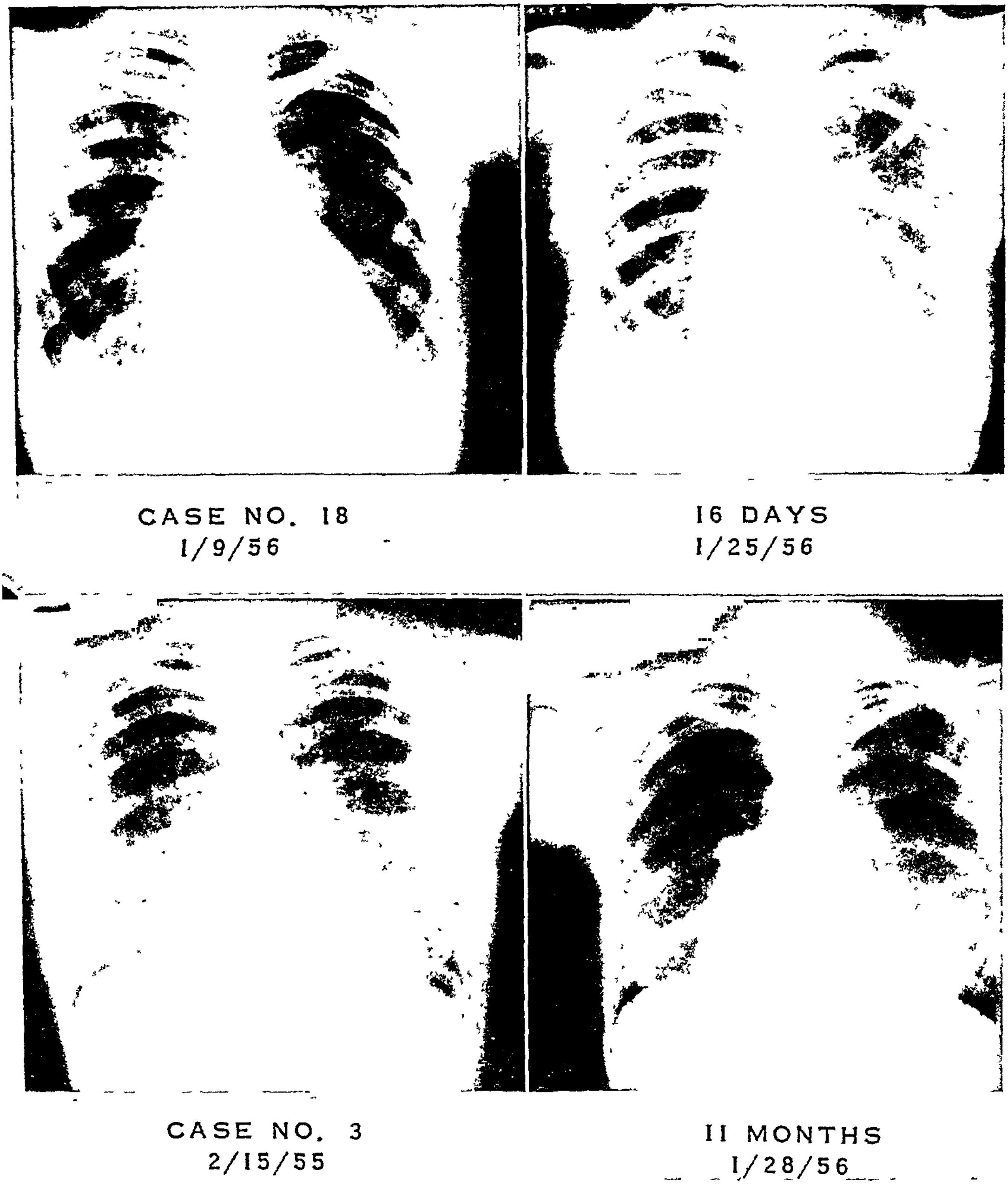
The obvious radiographic change in cardiac contour and diminution in size in the cardiac silhouette is reproduced in Fig. 12.

Pre- and postoperative purse-string pressure data within the left atrium are available in Table II. It will be noted that in most instances a highly satisfactory drop in pressure was obtained. The data in these living patients can conveniently be compared with similar data in those who did not survive. Examples of actual pressure tracings taken within the left atrium before and after annular constriction both in cases of relatively pure mitral insufficiency and in those complicated by concomitant mitral stenosis are shown in Figs. 11, 13, and 14.

## COMMENT

An honest and sincere effort has been made to present the entire picture of our initial four year effort to overcome surgically the detrimental effects of one of the commonest of valvular problems—mitral insufficiency. Much work and application lie ahead before one can categorically state that this procedure, or any other, materially affects the clinical course of patients so afflicted for the better. An attempt has been made to approach the problem in a most scientific and academic manner and the continuation of work on this project is to be kept at the same level. At least three aspects of the over all project need constant attention. (1) Can the technique be further improved to obtain a more satisfactory hemodynamic effect by either closed or open heart surgery or will the final solution await the development of some form of artificial valve? (2) Are the patients such as those in Group I whose ultimate results were so dismal, truly unsalvable? Continuing work in our experimental laboratory suggests another possible approach. Our laboratory associate, Dr. Domingos Moraes, is presently studying the effects of artificially produced chronic mitral insufficiency.[27] It is well known that the acute production of marked and significant mitral insufficiency in the laboratory animal is routinely attended by rapid death from fulminating pulmonary congestion

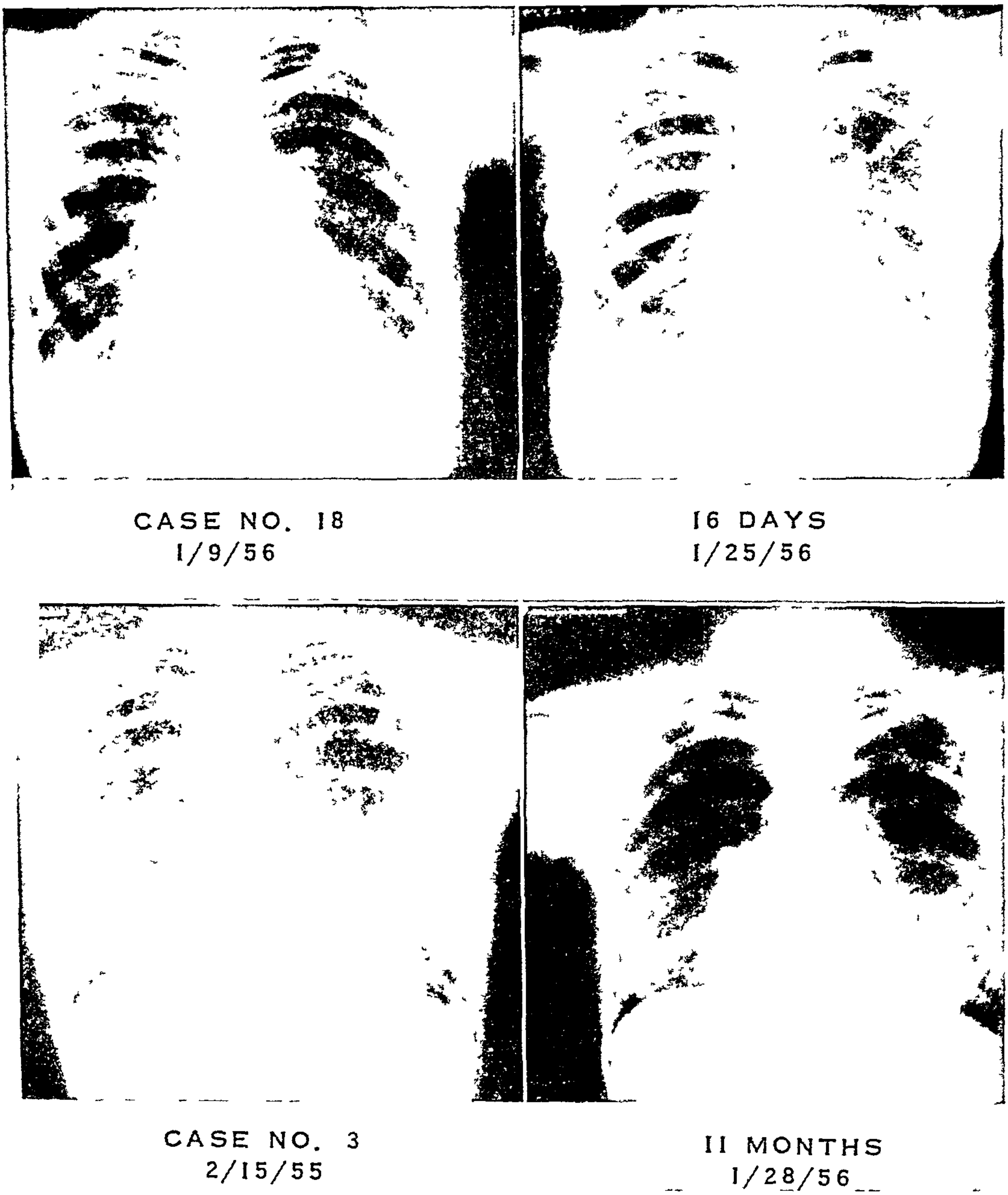
Therefore, to date no detailed reports of chronic mitral insufficiency are available in any quantity. Moraes has observed that when total ligation of the inferior vena cava below the renal veins is performed at the same operation as is the production of significant mitral insufficiency, approximately 50 per cent of the animals will survive. A considerable group of animals so prepared is presently being followed and the typical findings of clinical chronic mitral



A.

Fig 12.—*A* and *B*, Posteroanterior teleoroentgenograms of 4 cases which typify the changes noted. The decrease in size of the left ventricle as well as the relief of tension in the left atrium, rendering it quite flaccid, is obvious immediately at operation. This diminution in cardiac size is evident on the early postoperative x-rays and persists or improves further in the late postoperative periods.

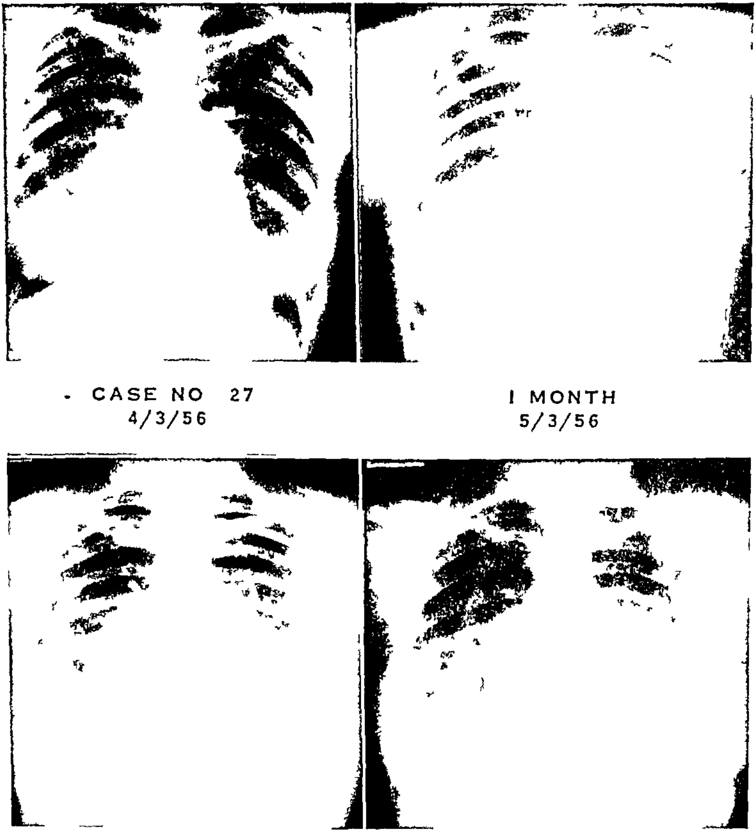
Therefore, to date no detailed reports of chronic mitral insufficiency are available in any quantity. Moraes has observed that when total ligation of the inferior vena cava below the renal veins is performed at the same operation as is the production of significant mitral insufficiency, approximately 50 per cent of the animals will survive. A considerable group of animals so prepared is presently being followed and the typical findings of clinical chronic mitral



*A*

Fig 12—*A* and *B*, Posteroanterior teleoroentgenograms of 4 cases which typify the changes noted. The decrease in size of the left ventricle as well as the relief of tension in the left atrium, rendering it quite flaccid, is obvious immediately at operation This diminution in cardiac size is evident on the early postoperative x-rays and persists or improves further in the late postoperative periods

insufficiency are being observed. A possible solution applicable to patients in intractable congestive failure resulting from mitral insufficiency might be the preliminary ligation of the vena cava. Presumably by temporarily reducing the return of blood to the right heart and pulmonary vascular bed such hearts may obtain a "breathing spell" and recover a measure of myocardial integrity. Mitral purse string then performed at a more optimum time conceivably may be better tolerated and result in an improved clinical state. (3) The unexplained death in Cases 6 and 7 at three and five months, respectively, when



CASE NO 27
4/3/56

I MONTH
5/3/56

CASE NO. I
1/13/55

I YEAR
1/28/56

*B*

Fig. 12 *B*. (*For legend see opposite pa*

these patients were enjoying better health (free from cardiac congestion), than they had known in years, need a solution. Possibly Case 24, who died rather suddenly on the fourth postoperative day, falls into this same category although in this instance it was not possible to improve satisfactorily left atrial dynamics which of course might explain her unexpected death. Whereas in none of the experimental animals, followed for many months, was there any evidence of a conduction defect presumably occasioned by trauma or distortion to the bundle of His, possibly some such complication as this may be involved. Why this type of reaction should occur at such a late date is inexplicable. In this regard transient atrioventricular block was observed during

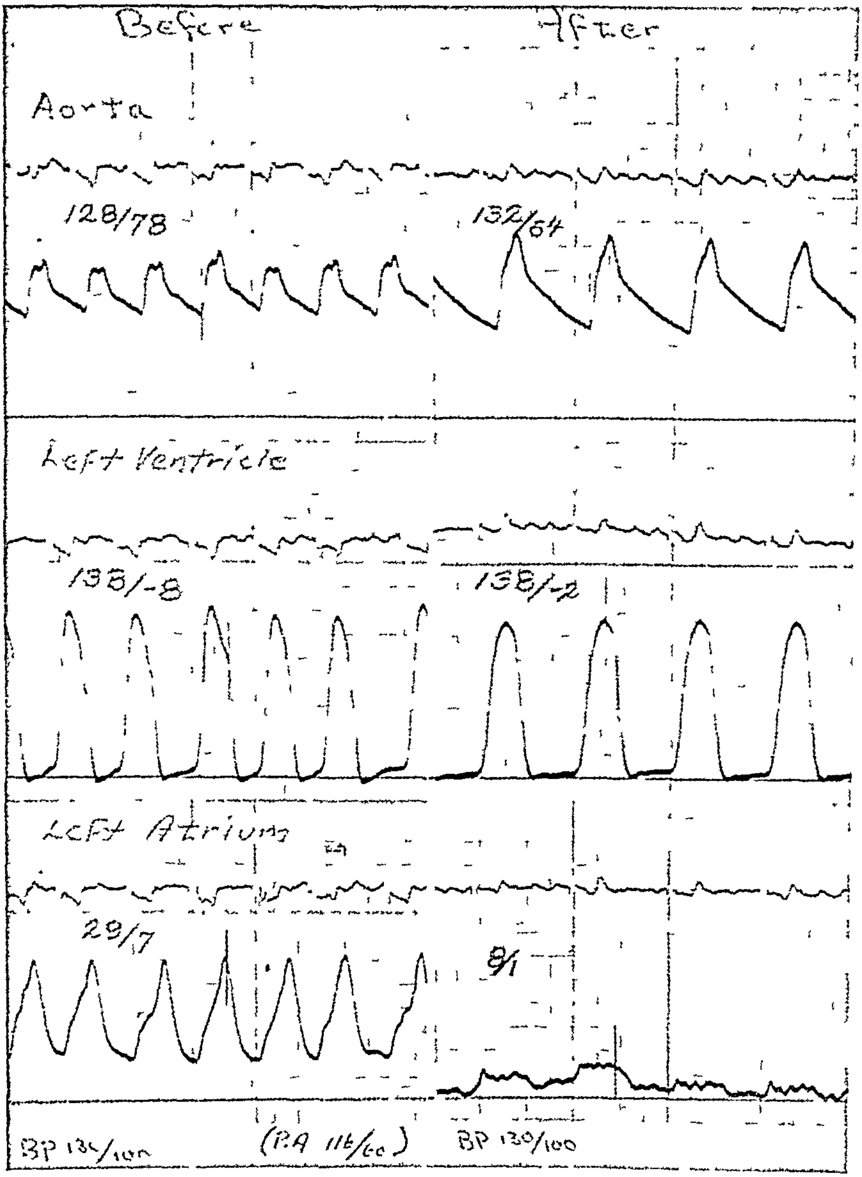


Fig. 13 —Tracings of aortic, left ventricular, and left atrial pressure pulses in Case 13, taken at surgery In those recorded before the procedure, note the left ventricular contour in the ejection phase Also note the typical regurgitant contour and pressure of the left atrial tracing The changes in all three pressure pulses are ideal in this instance

the postoperative period in 2 patients (Cases 17 and 19) but both cleared without sequelae. Case 11, who eventually died three months after surgery from subacute bacterial endocarditis, had an atrioventricular block both before and following surgery.

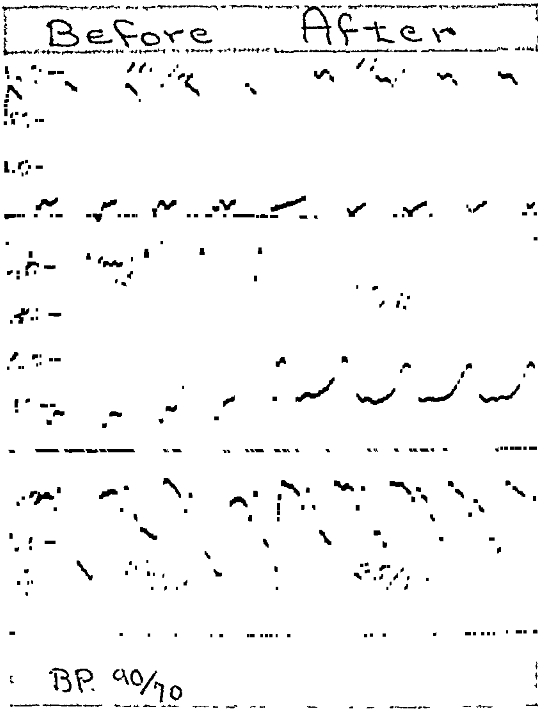


Fig 14.—These tracings were taken at surgery of Case 15, a 7-year-old child with pure, massive [illegible] he very satisfactory improvement in left atrial pressure pulse [illegible] this case, the question of rheumatic activity preoperatively [illegible] myocardial damage might preclude a good result. This patient, ho[illegible] well

SUMMARY AND CONCLUSIONS

1. The concept that mitral insufficiency is the result of three basic pathologic changes, namely, absolute loss of valve tissue, contraction and foreshortening of the chordae tendineae, and dilatation of the mitral annulus, was presented in detail.

2. For surgical correction of mitral insufficiency to be effective, each of these three factors must be taken into account.

3. Controlled circumferential constriction of the mitral annulus favorably affects each of these factors by reducing the size of the mitral orifice to be

occluded, by coapting the contracted and scarred but still pliable valve leaflets, and by providing relaxation to the stretched and distorted chordae tendineae.

4. Not only will total controlled circumferential constriction relieve in great measure existing mitral incompetence but, by permanent fixation of the atrioventricular annulus, it prevents further annular dilatation and subsequent progression of an insufficient state.

5. The technique for the proper performance of total annular constriction—mitral purse-string—has been detailed.

6. After three years of intensely applied experimental study during which time experimental mitral insufficiency, produced by every conceivable means, was proved to be correctable in routine manner, clinical application seemed justified.

7. Although ideally from a purely surgical viewpoint it would have been highly desirable to employ the procedure on those patients exhibiting more minor degrees of mitral valve incompetence in order to ensure a low operative mortality and a high percentage of living patients, this did not seem justified in the initial phase of clinical application. On the contrary, it was felt that early application should include only those patients showing far-advanced, even terminal, degrees of rheumatic heart disease with mitral insufficiency. Once the efficacy of the procedure was demonstrated on the rheumatic heart to simulate the findings observed on the experimental animal the operative procedure should then be applied at its proper level—the patient with early progressive mitral insufficiency.

8. A pilot group of 27 patients with rheumatic mitral insufficiency, pure or in combination with mitral stenosis, was subjected to mitral purse-string. In 6 a concomitant mitral commissurotomy was likewise performed.

9. Fourteen of these patients (Group I) were in intractable (terminal) failure with variable degrees of pulmonary congestion, hepatomegaly, ascites, and peripheral edema at the time of surgery.. Seven of these patients died within one month, 6 within eight months and one is living to date at three months. Despite the fact that in most instances mitral insufficiency had probably been corrected to a marked degree, pre-existing irreversible myocardial, pulmonary, and hepatic changes proved too great for the mere correction of the valve lesion to overcome. The fact however that mitral purse-string could be accomplished with accuracy under these conditions, that mitral stenosis was not produced, that no coronary damage ensued, that no erosion of the suture into the lumen of the left atrium occurred, that no additional myocardial damage was produced, that the procedure was tolerated in patients in so precarious a state, and that left atrial hemodynamics could be corrected, was ample evidence that the procedure deserves considerable merit and further trial on better patients indicated.

10. Thirteen other patients (Group II) in chronic congestive failure but temporarily correctable by medical means prior to surgery were likewise subjected to mitral purse-string. Three of these patients died within three months and two of these were due to avoidable technical errors. The remaining 10 patients are living in a vastly improved state from two to sixteen

months postoperative Hemodynamic, radiographic, and phonocardiographic objective evidence is available and has been presented to substantiate the obvious merit of the procedure

11 Further clinical trial in better patients in an earlier stage of their disease complex is not only warranted but mandatory

## REFERENCES

1 Davila J C, Mattson, W W, Jr, O'Neill T J E, and Glover, R P A Method for the Surgical Correction of Mitral Insufficiency (Preliminary Considerations) Surg, Gynec & Obst 98 407, 1954
2 Denton G R A Plastic Prosthesis (Without Moving Parts) for the Atrioventricular Valves, Surg Forum A C S, 1950, Philadelphia, 1951, W B Saunders Company
3 Benichoux, R, and Chalnot, P A Method for the Surgical Correction of Mitral Insufficiency J THORACIC SURG 30 148 1955
4 Bailey, C P Mitral and Aortic Insufficiency Henry Ford International Symposium on Cardiovascular Surgery, Philadelphia 1955, W B Saunders Company
5 Harken, D E, Black, H, Ellis L B, and Dexter, L The Surgical Correction of Mitral Insufficiency, J THORACIC SURG 28 604, 1954
6 Harken, D E Mitral Insufficiency The Henry Ford International Symposium on Cardiovascular Surgery, Philadelphia 1955, W B Saunders Company
7 Johns T N P, and Blalock, A Mitral Insufficiency The Experimental Use of a Mobile Polyvinyl Sponge Prosthesis, Ann Surg 71 465, 1955
8 Jordan, P, and Wible J Spring Valve for Mitral Insufficiency Preliminary Report, Arch Surg 71 468, 1955
9 Bailey, C P, Jamison W L Bakst, A E, Bolton, H E Nichols, H T, and Gemeinhardt, W The Surgical Correction of Mitral Insufficiency by the Use of Pericardial Grafts J THORACIC SURG 28 551, 1954
10 Glenn, W W L, Turk, L N, and Gentsch, T O The Implantation of Vascularized Grafts in the Chambers of the Heart An Experimental Approach to the Correction of Valvular Insufficiencies by Vertically Suspended Grafts, Surg Forum A C S, Philadelphia 1955, W B Saunders Company
11 Glover, R P Henderson A R, Margutti R, and Gregory, J The Fate of Intracardiac Pericardial Grafts, Surg Forum A C S, Philadelphia, 1953, W B Saunders Company
12 Logan, A, and Turner, R The Diagnosis of Mitral Incompetence Accompanying Mitral Stenosis, Review of 11 Cases Treated Surgically, Lancet 2 593, 1952
13 Murray, G, Wilkinson, F R, and McKenzie, R Reconstruction of the Valves of the Heart, Canad M A J 38 317, 1938
14 Moore, T C and Shumacker, H B Unsuitability of Transventricular Autogenous Slings for Diminishing Valvular Insufficiency, Surgery 33 173, 1953
15 Sakakibara, S Surgical Approach to Correction of Mitral Insufficiency, Ann Surg 142 196 1955
16 Templeton, J Y, and Gibbon, J H, Jr Experimental Reconstruction of the Valves of the Heart by Venous and Pericardial Grafts Ann Surg 129 161, 1949
17 Albanese, J C in discussion of Mitral Stenosis and Mitral Insufficiency Cardiovascular Surgery, Henry Ford Hospital, International Symposium, Philadelphia, 1955, W B Saunders Company, p 262
18 Davila, J C, Glover, R P Trout, R G, Mansure F, Wood, N Janton, O H and Iaia, B D Circumferential Suture of the Mitral Valve, J THORACIC SURG 30 531, 1955
19 Davila J C, in discussion of Mitral Stenosis and Mitral Insufficiency Cardiovascular Surgery, Henry Ford Hospital, International Symposium, Philadelphia, 1955, W B Saunders Company, p 263
20 Glover, R P, and Davila J C The Surgical Treatment of Mitral Insufficiency by Total Circumferential Suture of the Mitral Ring Circulation (In Press)
21 Davila, J C, Trout, R G, Sunner, J E, and Glover, R P A Simple Mechanical Pulse Duplicator for Cinematography of Cardiac Valves in Action, Ann Surg 143 4, 1956
22 Davila J C Moraes, D J, Glover, R P, et al Studies on the Production of Chronic Mitral Insufficiency and the Sequelae Thereof in Experimental Animals, In preparation for publication

*(For Discussion, see page 117)*

# MITRAL INSUFFICIENCY: TREATMENT BY POLAR CROSS-FUSION OF THE MITRAL ANNULUS FIBROSUS

HENRY T. NICHOLS, M.D. (BY INVITATION)
PHILADELPHIA, PA.

VARIOUS surgical attempts have been made in recent years to restore competence to the regurgitant mitral valve. These efforts have been directed in the main toward addition of tissue[1-5] or a prosthetic device[6-9] between the leaflets, suturing of one leaflet to the other,[10] distortion of the mitral annulus,[11-14] and elevation of the leaflets either by stretching,[15] cutting[11, 12] the chordae tendineae or insertion of a prosthetic beneath them.[8, 9] Each of these procedures represents an attempt to correct the pathologic entity or entities which to the respective reporter seemed to be responsible for the insufficiency. It is of interest to note that many of the basic principles and techniques being used today for correction of mitral insufficiency have been applied previously in attempts at the production of mitral stenosis in the animal laboratory.[16-18]

There are several pathologic entities such as congenital perforation of one or both leaflets, congenital cleft leaflets, and detachment of the chordae which give rise to mitral insufficiency, but which occur so infrequently that they deservedly have received little surgical attention.

There are three pathologic entities which now are generally accepted as being responsible for mitral insufficiency in the majority of cases: (1) retracted leaflets (Fig. 1, *A*), (2) dilated mitral annulus (Fig. 1, *B*), and (3) shortened chordae tendineae (Fig. 1, *C*). It is not uncommon to find all three to be present, and it may be difficult or impossible to determine which is predominant. Regardless of which of the three entities is present, absent, or predominant in a combination, failure of one leaflet to make contact with the other during systole always is evident.

The physiologic effect of mitral insufficiency is a reduction in the percentage of the left ventricular stroke output which enters the aorta. As the heart increases its rate in response to physical activity, its mechanical efficiency rapidly decreases in relation to the demand for increased aortic output because of the disproportionate reduction in the length of diastole (or left ventricular filling period) in the shortened cardiac cycle. This results in excessive fatigue which is relieved only by physical inactivity.

Since fatigue seems mild in contrast to the more dramatic episodes such as dyspnea, hemoptysis, and embolism which are seen in mitral stenosis, some physicians have been led erroneously to conclude that mitral insufficiency is

---

From the Department of Thoracic Surgery, Hahnemann Medical College and Hospital of Philadelphia, and The Bailey Thoracic Clinic, Philadelphia, Pa.

Read at the Thirty-sixth Annual Meeting of The American Association for Thoracic Surgery at Miami Beach, Fla., May 7 to 9, 1956.

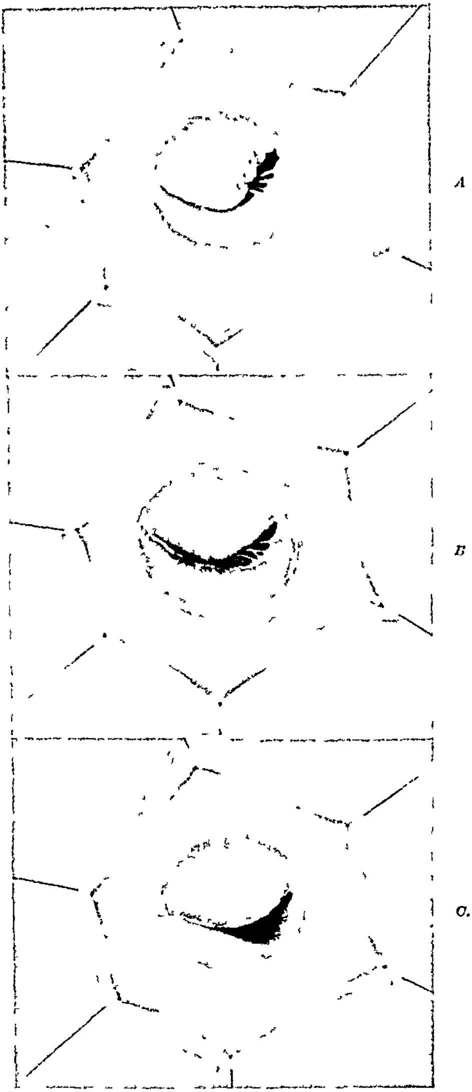


FIG. 1.—*A*, Intrinsic shortening and retraction of leaflet margins is the commonest cause of mitral insufficiency. *B*, Dilatation of the mitral annulus fibrosus causes incompetence most marked in the region of the posteromedial pole of the valve. *C*, Shortened chordae tendineae draw the margin of a leaflet downward into the ventricular chamber and systolic coaptation of the valve edges.

a benign condition. If the mitral insufficiency is great, the extra work load imposed upon the left ventricle eventually will lead to heart failure and death. Before this final event, the patient may be so restricted in physical activity that the joys of life seem few.

If the mitral insufficiency can be corrected, the mechanical efficiency of the left ventricle is increased and its over-all work load is decreased. The increased mechanical efficiency allows increased aortic outflow, especially during increased activity, lessens fatigue, and prevents or delays the onset of heart failure.

Attempts at introducing living tissue into the opening between the leaflets to block the regurgitation have resulted in a high operative mortality and frequently in disappointing end-results in the surviving patients. The application of prostheses into the same area likewise has eventuated unsatisfactorily.

These failures in attempts to block the opening between the leaflets have led to the development of various procedures designed to move one leaflet nearer the other thus enabling them to make contact during systole. Such operations have involved stretching and cutting shortened chordae to allow leaflets pulled down into the ventricle to rise into the atrium.[11-13] Prostheses have been placed beneath the mural leaflet without satisfaction.

We never have cut the chordae intentionally as we have been fearful of producing a flail leaflet. This, in itself, causes severe mitral insufficiency. Such a procedure would seem to have merit only in patients with shortened chordae and would seem contraindicated in those with retracted leaflets or dilated rings. Upward displacement of the mural leaflet has the same limitations in applicability.

Suturing of one leaflet to the other has been tried in an attempt to close the space between the two. We found that in some instances the leaflets were not strong enough to hold sutures under tension. Also, the inherent binding together of the edges of the leaflets by the pericardial sutures prevented descent of the edges into the ventricle and separation during diastole. If the leaflets were thin and pliable, this restriction was localized to the site of suturing. However, in those with thickened leaflets, the restraint was reflected throughout the leaflet edge and the entire cusp was limited in its movement with resultant stenosis.

More recently, our attention has been directed toward the annulus fibrosus and attempts have been made to displace one leaflet closer to the other so they might make contact during systole.

Hurwitt[19] suggested resection of portions of the annulus along with a wedge-shaped segment of ventricular myocardium. MacAllister[20] apparently was the first to attempt to constrict the mitral annulus fibrosus circumferentially in a clinical case. Davila and associates[13, 14] have applied a circumferential purse-string type suture around the entire mitral annulus.. Borrie[21] has done the same procedure, apparently entirely independently. Kay and Cross[11] have distorted the annulus in the region of the posteromedial commissure by placing sutures across the lumen of the atrium, through the annulus at either extremity, tying the free ends over buttons of cartilage outside the heart. Several modifications of this procedure to distort the annulus also have been made by Kay.[12]

The operation which we have named polar cross fusion is also an anatomic alteration of the mitral annulus. In the development of this procedure our objectives have been as follows: (1) to displace one leaflet closer to the other in the region of the incompetence so that their free margins may make contact during systole, (2) to place the approximating sutures around tissue that will hold under tension, (3) to avoid compromising normal leaflet action, (4) to avoid the production of physiologically significant mitral stenosis, and (5) to assure permanence of the anatomic alterations produced.

Our first attempt to accomplish the above objectives consisted of plication of the mitral annulus on the mural leaflet side by the placement of multiple interlocking mattress sutures within the substance of the annulus.[22] This shortened the annulus in this area and moved the mural leaflet closer to the septal leaflet sufficiently to correct regurgitations of small magnitude. However, it was not effective in abolishing the larger regurgitations. Also, this procedure was tedious and difficult technically because it required mobilization of the left circumflex coronary artery from its bed.

The above shortcomings led us next to consider plicating the annular ring by sutures placed between its mural and septal aspects in the region of the posteromedial commissure. In the animal laboratory, it was found that the sutures could be placed accurately and tied without great difficulty. Immediate and permanent fixation of the approximated portions of the ring was accomplished readily. We applied the polar cross fusion technique to our first patient on Aug. 16, 1955, with survival.

## TECHNIQUE

At the present time, the operation is performed through the left fifth intercostal space with the patient in the lateral position. If the fourth interspace is used, the exposure is limited for placement of the sutures. If the sixth intercostal space is used, there may be difficulty in relieving any coexisting mitral stenosis which might be present. The pericardium is opened by removing a 3 cm. wide strip of pericardium just posterior to and running parallel to the left phrenic nerve. This strip of pericardium is used to encase or ensheath the nonabsorbable suture to be placed inside the heart. The purse-stringed left atrial appendage is entered and exploration of the valve plus surgery for any existing mitral stenosis is carried out. The surgeon removes his right index finger from the atrium and moves to the opposite side of the operating table.

The ungloved left index finger is then introduced into the left atrium, the operator standing in front of the patient. A large curved needle swaged to a heavy Dacron suture is introduced through the left atrial wall at a point close to the incompetent valve pole (Fig. 2). If the valve is incompetent posteriorly, the intracardiac tip of the bare left index finger is used as a guide to direct the point of the needle under the septal aspect of the annulus in the region of the posterior pole of the valve. The needle point then is redirected to continue across the atrial lumen and to pass just beneath the annulus on the mural leaflet side. Finally it emerges from the left ventricular wall close to the atrioventricular junction. The point of a second needle swaged to the opposite end of the suture then is engaged into the substance of the Dacron suture itself (Fig. 2) near its entrance puncture through the left atrial wall in order to assure that it will pass into the left atrium through the same opening. Entrance through the same hole is important because it obviates the inadvertent inclusion of soft tissue within the loop of the suture when it is tied down. Necrosis of such included muscular tissue under pressure of the suture will tend to permit the approximated areas of the annulus to separate during the postoperative period.

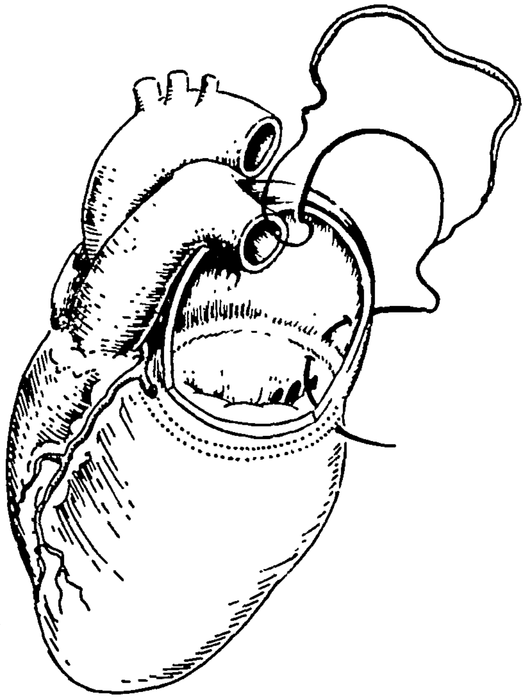


Fig. 2.

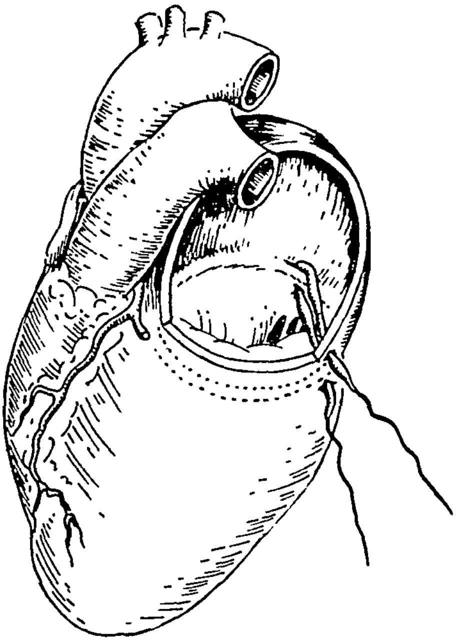


Fig. 3.

Fig. 2.—Placement of the plicating suture. One needle has pierced the left atrial wall, has been guided under the septal portion of the annulus fibrosus, has traversed the atrial lumen, and has passed under the mural portion of the ring to emerge from the left ventricular wall. The other needle point is imbedded temporarily within the Dacron suture in order to enter the atrial chamber through the same puncture. The pericardial sheath has been affixed to the mid-portion of the suture.

Fig. 3.—After the second needle enters the atrial chamber, it is caused to emerge close to the first but at a level just above (superficial to) the mural portion of the annulus.

After the point of the second needle is passed through the same atrial puncture site, it is disengaged from the Dacron suture merely by applying traction to the end of the suture which already has been passed through the heart. The needle point then is directed across the lumen of the left atrium to pass just above the annulus on the mural leaflet side and then to emerge from the left ventricular wall in close proximity to the other. The suture then is adjusted, by traction on its ends, so that the part remaining within the heart is completely encased by the homograft pericardium (Fig. 3). The distal ends of the pericardial strip are clamped and then freed from the Dacron by cutting the attaching arterial silk stitches. The mobilized suture then is tied outside the heart, making certain, by use of the left index finger within the heart, that the two points of the annulus enclosed within the loop of the suture are brought into firm apposition (Fig. 4). Since there usually is a small amount of ventricular muscle caught within the loop of the suture between the knot and the underlying mural part of the ring, we have waited several minutes after making the first throw in the knot to allow such muscle to give way under the tension of the suture. The first throw is then tightened again, and this is followed by completion of several additional throws in the knot. A fine cotton suture passed through the substance of the Dacron tends to ensure tightness of the knot. The emerging ends of the pericardial strip are approximated or tied together and this apposition is rendered permanent by placing several cotton stitches through them.

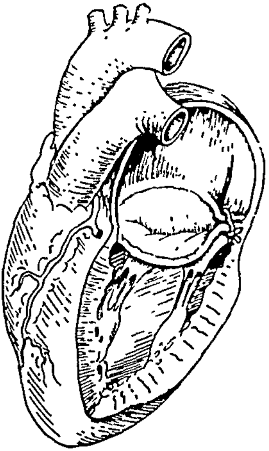


Fig 4.

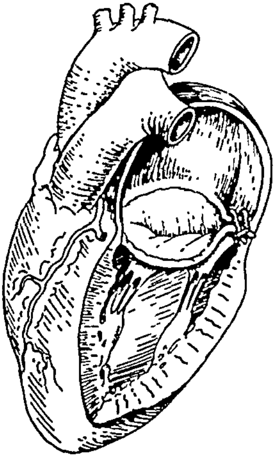


Fig. 5.

Fig. 4.—The suture is tied down as described in the text after the pericardial sheath has been separated from the Dacron in the region of the knot. At this point competence is restored to the valve in most instances.

Fig. 5.—In some individuals a second such plicating suture is necessary in order to overcome the entire insufficiency.

A second suture is placed alongside the first (Fig. 5), if it is necessary for the complete abolition of the regurgitant jet. At the present time, we are uncertain whether a second suture should not always be placed to act as a buffer to decrease the tension on the first one.

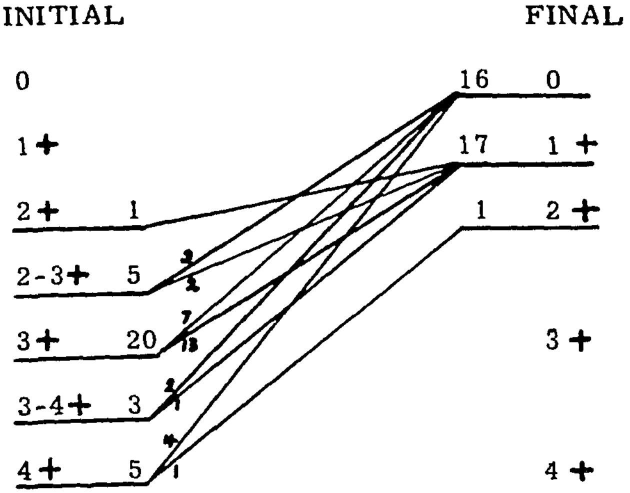
Cross-fusion of the annulus at the anterior pole of the valve can be selectively carried out just as is described for application at the posterior commissure in those few patients in whom the leak lies anteriorly.

## DISCUSSION

The effectiveness of this procedure in blocking the regurgitation at the time of surgery has been most dramatic (Table I). Among the 34 patients operated on, the regurgitation no longer was palpable and apparently was abolished completely in sixteen instances. In 17 patients, there remained a palpable regurgitant jet which had been reduced to an estimated 1 plus magnitude. Many of these were just barely palpable to the ungloved finger, but if any regurgitation at all could be felt, it was recorded as of 1 plus magnitude. In only one patient was a 2 plus regurgitation permitted to remain. This was an extremely ill woman in whom one suture reduced the insufficiency from 4 plus to 2 plus, and it was felt best not to attempt placement of a second suture because of her extremely poor condition on the operating table. In the majority of these individuals, effectiveness of the correction at the time of surgery was evidenced by the lowering of tension within the left atrium and, in some, this was further attested to by a sudden and obvious decrease in left ventricular size.

Table I

ESTIMATED M.I. AT SURGERY



We have been encouraged to feel that we are accomplishing the objectives previously laid down. The bases of the leaflets certainly are brought into closer proximity so that their free margins are enabled to make contact during systole, thus effectively blocking the regurgitation.

The mitral annulus fibrosus seems to satisfy the requirement of a substance strong enough to hold sutures under tension. At least it is the only tendonlike structure available. The atrial wall and ventricular wall are muscular, and the incapacity of muscle tissue in holding sutures under tension is well recognized. The leaflets themselves will not hold sutures unless heavily fibrosed and, if such fibrosis is present, the creation of mitral stenosis is the price of correcting the regurgitation by this method. Polar cross-fusion of the annulus does not compromise leaflet action because only the leaflet's anchorage point the mitral annulus is enclosed within the loop of the suture. The leaflets themselves remain capable of swinging with the alternations of the hydrodynamic stresses during the cardiac cycle. Physiologic mitral stenosis is not produced because the cross-sectional area of the atrioventricular passageway at the valve annulus is considerably greater than at the leaflet orifice. Permanent fusion of the plicated portions of the annulus should be assured by the use of the heavy nonabsorbable suture (80 pound test braided Dacron) plus the ensheathment of autogenous pericardium. The Dacron suture is used primarily for its bulk rather than its tensile strength in order to obviate any tendency of the suture to cut through substance of the annulus. The pericardial sheath around the suture likewise buffers any tendency toward cutting through. Ultimate conversion of the autogenous pericardium into dense scar tissue assures a fibrous coupling of the approximated areas of annulus (Figs. 6A and 6B).

The mortality (Table II) experienced by the first 34 patients operated on was gratifyingly lower than that of some of the procedures which we have performed in the past and some which have been and presently are being done by others. The very fact that no deaths have occurred on the operating table has convinced us that the procedure is far safer than others we have tried. We have had to treat fibrillation once during surgery, correction being accomplished after the plicating suture was tied down.

TABLE II

| | |
|---|---|
| Total cases (polar cross fusion) | 34 |
| Concomitant mitral commissurotomy | 9 |
| Associated A. S. A. I. (aortic lesion not corrected) | 2 |
| Deaths to date | 5 (14.7%) |

The causes of death (Table III) lead us to believe that the mortality can be lowered in the future. Certainly the technical error is avoidable, and the basal skull fracture was not related to the operative procedure. The operation was completed in the patient with severe hypotension only because we felt that to retreat without giving mechanical relief to the heart assuredly would lead to fatality. Resistant strains of bacteria seem to be on the increase, but fortunately newer antibiotics also continue to appear. Selection of patients can, in some instances, only be settled by the philosophy of the patient, cardiologist and surgeon in undertaking calculated risks.

TABLE III. CAUSES OF DEATH

1. Technical error (occlusion of right coronary artery)
2. Improper selection (incorrect evaluation of co-existent aortic stenosis)
3. Severe hypotension (during thoracotomy)
4. Basal skull fracture due to fall (13 days postop.)
5. Acute bacterial endocarditis

Any attempt at evaluation of the results of surgery with reference to the level of physical activity would not be valid at this time. None of the patients as yet is one year postoperative. The majority of the patients have been

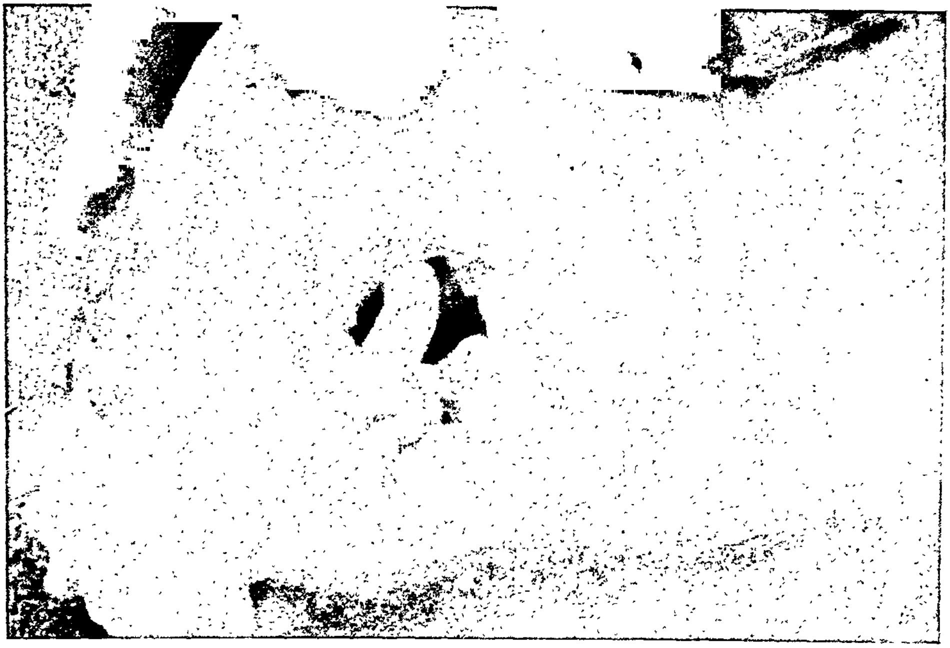


Fig. 6A.—Photograph of a mitral valve specimen showing a pericardial hammock which had been placed three years previously.

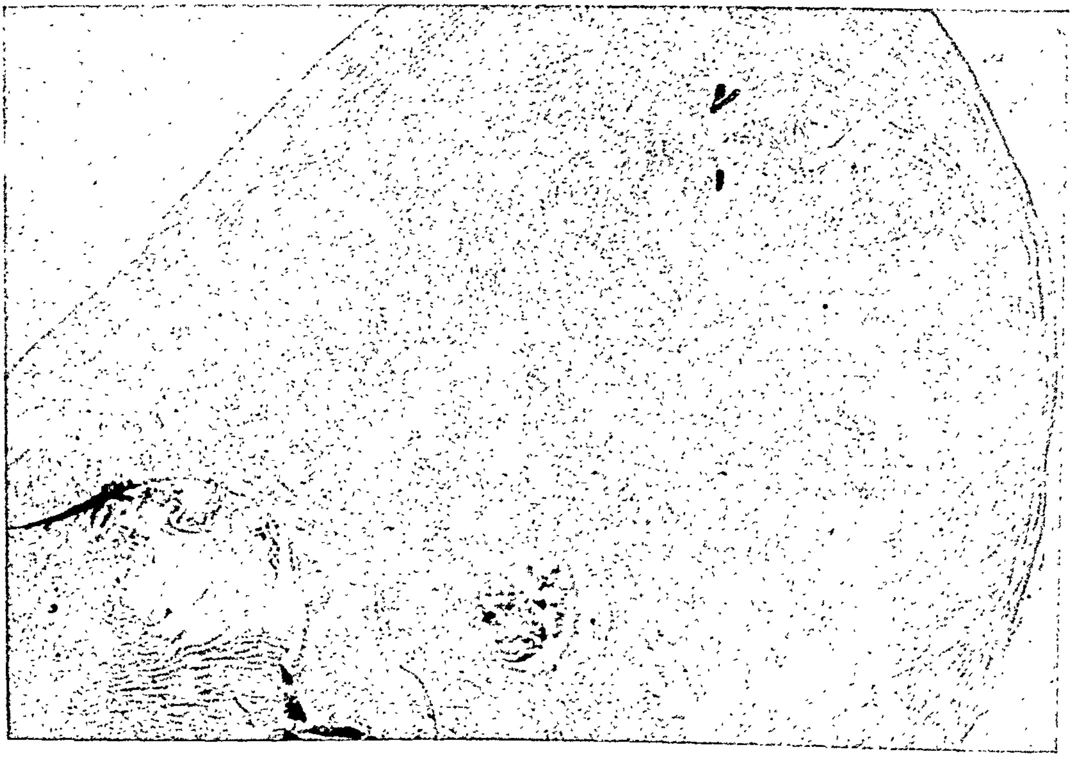


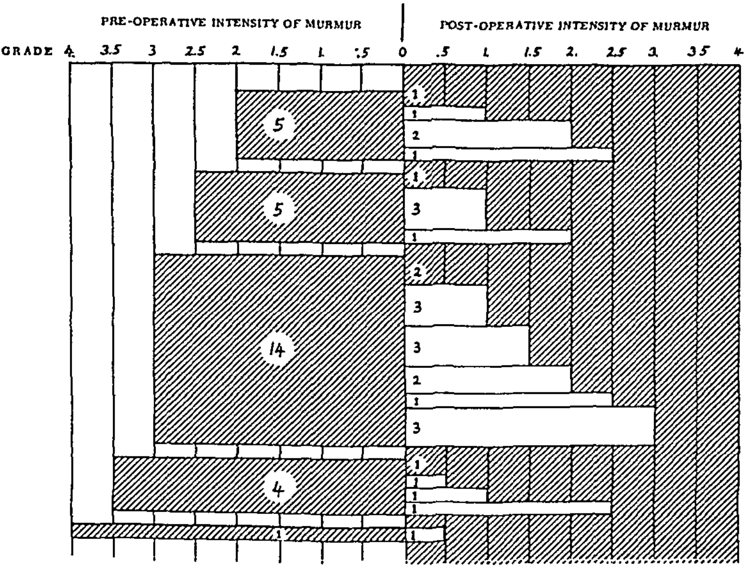
Fig. 6B.—Photomicrograph of cross-section through fibrosed pericardial hammock showing collagenous metaplasia.

operated on within the past five months, and have not, as yet, tested their maximal exertional ability. An evaluation of their status will be reported at a later date.

The fact that there is uncertain correlation between the intensity of a systolic murmur and the estimated amount of mitral regurgitation found at surgery is appreciated universally. The fact that no regurgitation is found in some patients with systolic murmurs, and that, in other patients, regurgitation is found, when no murmur was detected preoperatively, serves to demonstrate the impossibility of a true auscultatory correlation. However, we felt that a comparison of preoperative and postoperative murmurs might be of some usefulness and such is, therefore, presented in Table IV. Fig. 7 shows phonocardiographic studies in a patient in whom the systolic murmur was completely abolished.

TABLE IV

CHANGES IN SYSTOLIC MURMUR



NUMBERS IN BOXES REFER TO NUMBER OF CASES

We have performed left heart catheterization by the method of Björk[22] and Kent and associates[23] in 21 patients preoperatively and, to date, in 8 cases postoperatively. In none of the postoperative tracings has there been evidence that mitral stenosis was produced by surgery. The typical findings in the left atrial curves have been similar to those shown in Fig. 8 in which the enlarged V wave has been altered markedly and in which the mean atrial pressure has been reduced.

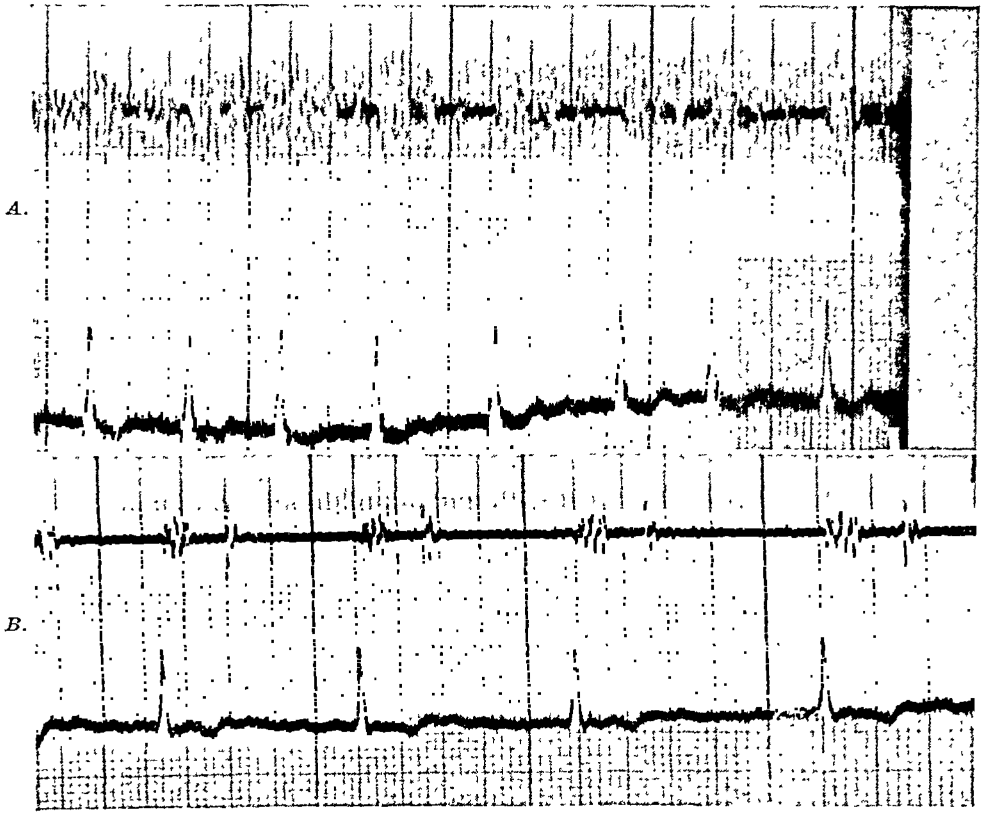


Fig. 7.—*A*, Preoperative phonocardiographic tracing indicative of long loud systolic murmur.

*B*, Postoperative phonocardiographic tracing in the same patient showing practically complete abolition of systolic murmur.

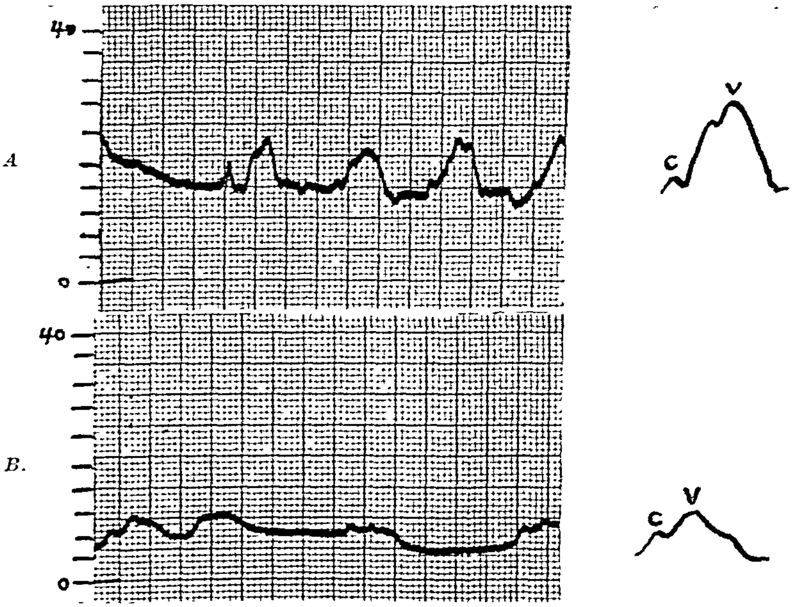


Fig. 8.—*A*, Preoperative pressure tracing obtained from the left atrium during left heart catheterization (Björk method). Note the large V wave suggestive of mitral insufficiency.

*B*, Postoperative pressure tracing obtained from the same patient showing normal C-V wave.

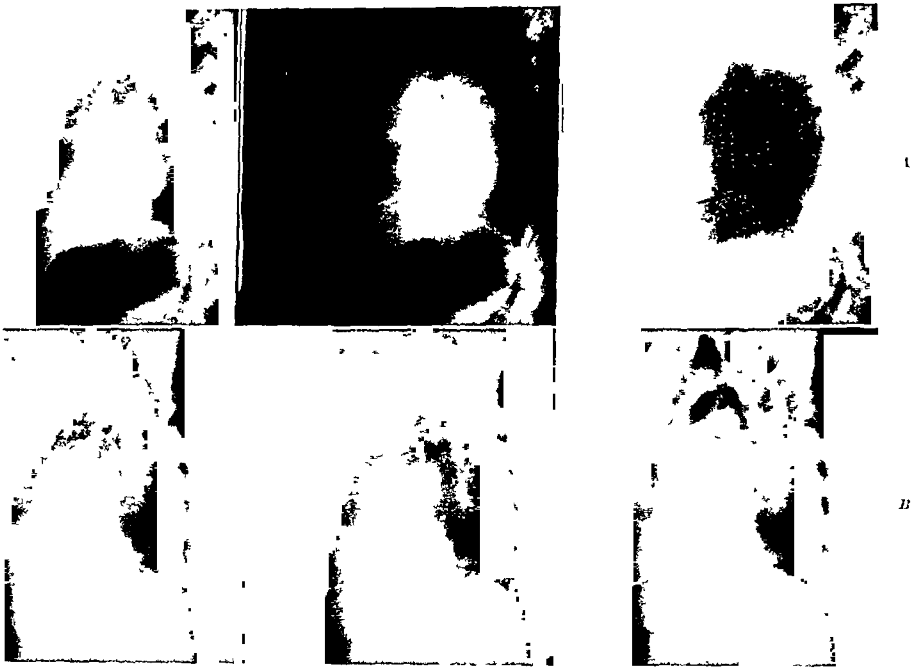
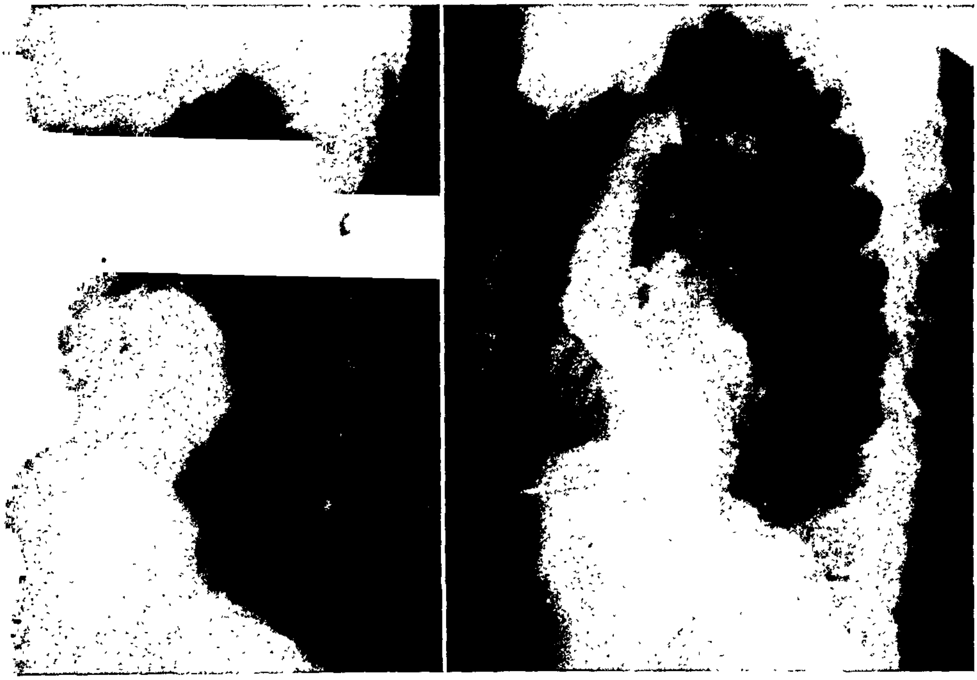


Fig. 9.—A, F, F. Serial radiograms taken respectively (left-to-right, April 10, 1956) just before, during, and immediately *after direct injection of 50 c.c. of 70 per cent Diodrast into the left* ventricular chamber (Smith technique). Note opacification of the left atrium indicating regurgitation.

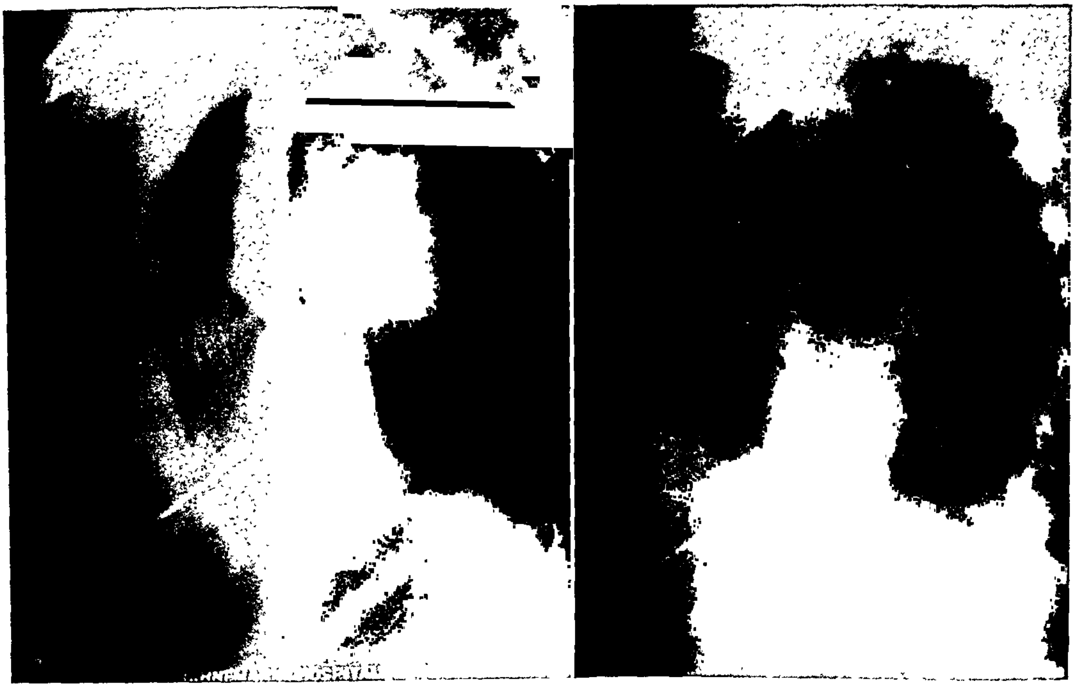
*B*. Similar ventriculograms taken in the same patient three weeks (May 2, 1956) after polar cross fusion of the annulus. Note marked reduction in the extent of opacification of the left atrium.



A. B.

Fig. 10.—*A*, Preoperative ventriculogram obtained on Patient J. A., a 21-year-old white woman. Note opacification of the left atrium indicating an extremely incompetent mitral valve.

*B*, Ventriculogram obtained on J. A. three weeks after performance of polar cross-fusion of the annulus for a 3 plus incompetence of the posteromedial pole of the valve. Note the great reduction of opacification of the left atrium which is indicative of a similar reduction in the mitral insufficiency.



A. B.

Fig. 11.—*A*, Preoperative ventriculogram obtained on Patient M. S., a 21-year-old white woman. Note extreme opacification of left atrium.

*B*, Ventriculogram obtained on the same patient two and one-half weeks after polar cross-fusion of the posteromedial pole of the valve for a 3 plus insufficiency. Note the near-absence of opacification of the left atrium indicating the great degree of restoration of mitral competence which has been achieved.

Left cardiac ventriculogram by direct needle puncture into the left ventricle as advocated by Beato Nuñez,[24] and Smith and associates[26-28] has been performed in 19 patients preoperatively, and in 6 postoperatively. Some of the postoperative ventriculograms have shown no demonstrable dye entering the left atrium, while the others have shown a decreased amount in comparison with the preoperative ventriculogram. Figs. 9, 10, and 11 demonstrate comparative pre- and postoperative ventriculograms.

Decrease in heart size has been demonstrated in some of the postoperative patients. Fig. 12 shows such decrease Others have shown slight reduction in size. No alteration has been seen as yet in the remainder. To evaluate the ultimate changes in heart size will require a longer follow-up period.

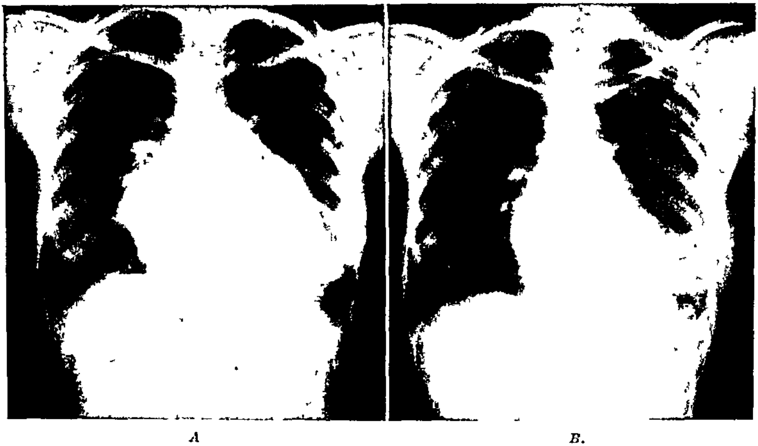


Fig 12—*A*, Preoperative 72-inch posteroanterior roentgenogram of L S, a 31-year-old white woman with severe (3 plus) mitral insufficiency

*B*, Similar roentgenogram obtained 3 weeks after polar cross-fusion of the mitral valve The great reduction in heart size suggests improvement in cardiac function

## SUMMARY AND CONCLUSIONS

The surgical technique for establishing polar cross-fusion in mitral insufficiency has been presented. The objectives of the operation and the methods of accomplishing them are presented. The operation has been used successfully in valves showing the three most common pathologic deformities, shortened or retracted leaflet, dilated ring, and shortened chordae tendineae. We have used it in 2 cases in which there were no palpable chordae attached to the mural leaflet. This lack of support permitted this leaflet to override the septal leaflet.

In one of our 34 patients, the procedure was applied across the anterior pole of the valve only, as this was the region of predominant regurgitation.

In another, it was applied across the area of the anterolateral commissure in addition to the posteromedial commissural area. In the remaining patients, the procedure was carried out only across the posterior pole of the valve where the vast majority of insufficiencies occur.

Obviously, polar cross-fusion of the annulus cannot be helpful in an extremely rigid or calcified valve.

We have no experience with the procedure in repairing insufficiency created surgically during the performance of mitral commissurotomy. In cases in which the chordae have been severed or the leaflets inadverently torn or cut, it is doubtful if it will be of as great value as in those lesions which are produced by the rheumatic process. It may well prove of value, however, in those instances in which the division of the commissure has been done accurately with respect to the leaflets but in which the incision has been allowed to continue on through the mitral ring. It may be possible in these individuals to restore the ring integrity by polar cross-fusion.

## REFERENCES

1. Bailey, C. P., O'Neill, J. T. E., Glover, R. P., Jamison, W. L., and Redondo-Ramirez, H. P.: Surgical Repair of Mitral Insufficiency (A Preliminary Report), Dis. Chest 19: 125, 1951.
2. Glenn, W. W. L., and Turk, L. N.: Implantation of Vascularized Grafts in the Chambers of the Heart. An Experimental Approach to the Correction of Valvular Insufficiencies, Surg. Forum American College of Surgeons, Atlantic City, N. J., Nov. 15, 1954.
3. Bailey, C. P., Jamison, W. L., Bakst, A. E., Bolton, H. E., Nichols, H. T., and Gemeinhardt, W.: The Surgical Correction of Mitral Insufficiency by the Use of Pericardial Grafts, J. Thoracic Surg. 28: 551, 1954.
4. Murray, G., Wilkenson, L. R., and MacKenzie, R.: Reconstruction of the Valve of the Heart, Canad. M. A. J. 38: 317, 1938.
5. Cleland, W.: Personal communication to Charles P. Bailey, 1952.
6. Harken, D. E., Black, H., Ellis, L. B., and Dexter, L.: The Surgical Correction of Mitral Insufficiency, J. Thoracic Surg. 28: 604, 1954.
7. Blalock, A., and Johns, T. N. P.: Mitral Insufficiency: The Experimental Use of Mobile Polyvinyl Sponge Prosthesis, Ann. Surg. 140: 333, 1954.
8. Carter, M. G., Gould, J. M., and Mann, B. F.: Surgical Treatment of Mitral Insufficiency, J. Thoracic Surg. 26: 574, 1953.
9. Benichoux, R., and Chalnot, P.: A Method for the Surgical Correction of Mitral Insufficiency, J. Thoracic Surg. 30: 148, 1955.
10. Bailey, C. P., Bolton, H. E., and Redondo-Ramirez, H. P.: Surgery of the Mitral Valve, Surg. Clin. North America 32: 1807, 1952.
11. Kay, E. B., and Cross, F. S.: Surgical Treatment of Mitral Insufficiency, J. Thoracic Surg. 29: 618, 1955.
12. Kay, E. B., and Cross, F. S.: Surgical Treatment of Mitral Insufficiency, Surgery 37: 697, 1955.
13. Davila, J. C., Mattson, W. W., Jr., O'Neill, J. T. E., and Glover, R. P.: A Method for Surgical Correction of Mitral Insufficiency, Surg., Gynec. & Obst. 98: 407, 1954.
14. Davila, J. C., Glover, R. P., Trout, R. G., Mansure, F. S., Wood, N. E., Janton, O. H., and Ioia, B. D.: Circumferential Suture of the Mitral Ring, J. Thoracic Surg. 30: 531, 1955.
15. Dodrill, F. D.: Experience With the Mechanical Heart, J. A. M. A. 154: 299, 1954.
16. Bernheim, B. M.: Experimental Surgery of the Mitral Valve, Bull. Johns Hopkins Hosp. 20: 107, 1909.
17. Wilson, W. C.: Studies in Experimental Mitral Obstruction in Relation to Surgical Treatment of Mitral Stenosis, Brit. J. Surg. 18: 259, 1930.
18. Ellison, R. G., Major, R. C., Pickering, R. W., and Hamilton, W. F.: Technique of Producing Mitral Stenosis of Controlled Degree, J. Thoracic Surg. 24: 154, 1952.
19. Hurwitt, E. S.: Experimental Production and Correction of Mitral Insufficiency, Surgery 37: 15-31, 1955.

20. MacAllister, F.: Personal communication to C. P. Bailey.
21. Borrie, J.: Mitral Insufficiency, J. THORACIC SURG. 30: 687, 1955.
22. Björk, V. O.: Direct Pressure Measurements in the Left Atrium, the Left Ventricle, and the Aorta, Acta chir. Scandinav. 107: 466, 1954.
23. Kent, E. M., Ford, W. B., Fisher, D. L., and Childs, T. B.: The Estimation of the Severity of Mitral Regurgitation, Ann. Surg. 141: 47, 1955.
24. Beato Nuñez, V., and Ponsdomenech, E. R.: Heart Puncture; Cardioangiography: Clinical and Electrocardiographic Results, Am. Heart J. 41: 855, 1951.
25. Smith, P. W., Wilson, C. W., Cregg, H. A., and Klassen, K. P.: Cardioangiography, J. THORACIC SURG. 28: 273, 1954.
26. Cregg, H. A., Smith, P. W., Wilson, C. W., and Bull, J. W.: Cardioangiography, Radiology 65: 368, 1955.
27. See Reference No. 25.

## DISCUSSION

## (PAPERS BY GLOVER, AND DAVILA; AND NICHOLS)

DR. MELVIN NEWMAN, Brooklyn, N. Y.—Our group at the State University of New York in Brooklyn believe that the operation of the future will be the complete replacement of the mitral valve by a plastic prosthesis.

(slide) This simple ball valve has been used for complete replacement of the mitral valve in 25 dogs. The features of the operative technique are first, complete by-pass of the circulation by a pump oxygenator; second, ventricular fibrillation produced by electric shock, and this completely obviates the problem of air embolism; and third, insertion and fixation of the valve under direct vision through a left atrial approach. Ventricular fibrillation has been promptly reversible in all dogs and in the 2 patients on whom operative correction of interventricular septal defects has been done. The ball valve, as shown, has permitted survival for a maximum of four months and is unsuitable for clinical use because of thrombus formation, emboli, imperfections in fixation technique. However, the operative approach can be applied to any type of mitral valve surgery which may be done in the future.

DR. HARRISON BLACK, Boston, Mass.—I have enjoyed very much the presentations of Drs. Glover and Nichols, and their important contributions to this subject. On Dr. Harken's services in Boston we have wrestled with this difficult problem over the years. It is apparent that, although some patients have been greatly helped, the ultimate salvage when they are followed for a significant time leaves one dissatisfied with all the techniques currently available including intra-auricular baffles, annulus distortion, external annulus constriction, and external bolstering of the posterior herniation of the valve complex. At the risk of seeming repetitious, however, I believe I should re-emphasize several principles which constitute a distillate of our experience in dealing with this problem.

First of all, as we have been reminded by the authors, it is extremely important not to immobilize the leaflets. Any procedure which does this, as we have stressed on numerous occasions in the past, is going to result in damaging obstruction to forward flow. We cannot trade stenosis for insufficiency.

Second, the aneurysmal dilatation of the left auricle which occurs in the advanced stages of this disease must be corrected. Patients with predominant mitral insufficiency do not become significantly symptomatic, for the most part, until an aneurysmal dilatation of the left auricle has occurred and left ventricular failure has supervened. As the systolic jet weakens the auricular wall the chamber enlarges to enormous size and the pull of gravity in the erect position causes it to fall posteriorly toward the diaphragm. This in turn results in a sliding hernia of the valve leaflets at the posterior commissure which aggravates their incompetence by pulling them further apart. It seems reasonable

In another, it was applied across the area of the anterolateral commissure in addition to the posteromedial commissural area. In the remaining patients, the procedure was carried out only across the posterior pole of the valve where the vast majority of insufficiencies occur.

Obviously, polar cross-fusion of the annulus cannot be helpful in an extremely rigid or calcified valve.

We have no experience with the procedure in repairing insufficiency created surgically during the performance of mitral commissurotomy. In cases in which the chordae have been severed or the leaflets inadverently torn or cut, it is doubtful if it will be of as great value as in those lesions which are produced by the rheumatic process. It may well prove of value, however, in those instances in which the division of the commissure has been done accurately with respect to the leaflets but in which the incision has been allowed to continue on through the mitral ring. It may be possible in these individuals to restore the ring integrity by polar cross-fusion.

## REFERENCES

1. Bailey, C. P., O'Neill, J. T. E., Glover, R. P., Jamison, W. L., and Redondo-Ramirez, H. P.: Surgical Repair of Mitral Insufficiency (A Preliminary Report), Dis. Chest 19: 125, 1951.
2. Glenn, W. W. L., and Turk, L. N.: Implantation of Vascularized Grafts in the Chambers of the Heart. An Experimental Approach to the Correction of Valvular Insufficiencies, Surg. Forum American College of Surgeons, Atlantic City, N. J., Nov. 15, 1954.
3. Bailey, C. P., Jamison, W. L., Bakst, A. E., Bolton, H. E., Nichols, H. T., and Gemeinhardt, W.: The Surgical Correction of Mitral Insufficiency by the Use of Pericardial Grafts, J. THORACIC SURG. 28: 551, 1954.
4. Murray, G., Wilkenson, L. R., and MacKenzie, R.: Reconstruction of the Valve of the Heart, Canad. M. A. J. 38: 317, 1938.
5. Cleland, W.: Personal communication to Charles P. Bailey, 1952.
6. Harken, D. E., Black, H., Ellis, L. B., and Dexter, L.: The Surgical Correction of Mitral Insufficiency, J. THORACIC SURG. 28: 604, 1954.
7. Blalock, A., and Johns, T. N. P.: Mitral Insufficiency: The Experimental Use of Mobile Polyvinyl Sponge Prosthesis, Ann. Surg. 140: 333, 1954.
8. Carter, M. G., Gould, J. M., and Mann, B. F.: Surgical Treatment of Mitral Insufficiency, J. THORACIC SURG. 26: 574, 1953.
9. Benichoux, R., and Chalnot, P.: A Method for the Surgical Correction of Mitral Insufficiency, J. THORACIC SURG. 30: 148, 1955.
10. Bailey, C. P., Bolton, H. E., and Redondo-Ramirez, H. P.: Surgery of the Mitral Valve, Surg. Clin. North America 32: 1807, 1952.
11. Kay, E. B., and Cross, F. S.: Surgical Treatment of Mitral Insufficiency, J. THORACIC SURG. 29: 618, 1955.
12. Kay, E. B., and Cross, F. S.: Surgical Treatment of Mitral Insufficiency, Surgery 37: 697, 1955.
13. Davila, J. C., Mattson, W. W., Jr., O'Neill, J. T. E., and Glover, R. P.: A Method for Surgical Correction of Mitral Insufficiency, Surg., Gynec. & Obst. 98: 407, 1954.
14. Davila, J. C., Glover, R. P., Trout, R. G., Mansure, F. S., Wood, N. E., Janton, O. H., and Ioia, B. D.: Circumferential Suture of the Mitral Ring, J. THORACIC SURG. 30: 531, 1955.
15. Dodrill, F. D.: Experience With the Mechanical Heart, J. A. M. A. 154: 299, 1954.
16. Bernheim, B. M.: Experimental Surgery of the Mitral Valve, Bull. Johns Hopkins Hosp. 20: 107, 1909.
17. Wilson, W. C.: Studies in Experimental Mitral Obstruction in Relation to Surgical Treatment of Mitral Stenosis, Brit. J. Surg. 18: 259, 1930.
18. Ellison, R. G., Major, R. C., Pickering, R. W., and Hamilton, W. F.: Technique of Producing Mitral Stenosis of Controlled Degree, J. THORACIC SURG. 24: 154, 1952.
19. Hurwitt, E. S.: Experimental Production and Correction of Mitral Insufficiency, Surgery 37: 15-31, 1955.

20. MacAllister, F.: Personal communication to C. P. Bailey.
21. Borrie, J.: Mitral Insufficiency, J. Thoracic Surg. 30: 687, 1955.
22. Bjork, V. O.: Direct Pressure Measurements in the Left Atrium, the Left Ventricle, and the Aorta, Acta chir. Scandinav. 107: 466, 1954.
23. Kent, E. M., Ford, W. B., Fisher, D. L., and Childs, T. B.: The Estimation of the Severity of Mitral Regurgitation, Ann. Surg. 141: 47, 1955.
24. Beato Nuñez, V., and Ponsdomenech, E. R.: Heart Puncture, Cardioangiography: Clinical and Electrocardiographic Results, Am. Heart J. 41: 855, 1951.
25. Smith, P. W., Wilson, C. W., Gregg, H. A., and Klassen, K. P.: Cardioangiography, J. Thoracic Surg. 28: 273, 1954.
26. Gregg, H. A., Smith, P. W., Wilson, C. W., and Bull, J. W.: Cardioangiography, Radiology 65: 368, 1955.
27. See Reference No. 25.

## DISCUSSION

## (PAPERS BY GLOVER, AND DAVILA, AND NICHOLS)

DR. MELVIN NEWMAN, Brooklyn, N. Y.—Our group at the State University of New York in Brooklyn believe that the operation of the future will be the complete replacement of the mitral valve by a plastic prosthesis.

(slide) This simple ball valve has been used for complete replacement of the mitral valve in 25 dogs. The features of the operative technique are first, complete by-pass of the circulation by a pump oxygenator, second, ventricular fibrillation produced by electric shock, and this completely obviates the problem of air embolism; and third, insertion and fixation of the valve under direct vision through a left atrial approach. Ventricular fibrillation has been promptly reversible in all dogs and in the 2 patients on whom operative correction of interventricular septal defects has been done. The ball valve, as shown, has permitted survival for a maximum of four months and is unsuitable for clinical use because of thrombus formation, emboli, imperfections in fixation technique. However, the operative approach can be applied to any type of mitral valve surgery which may be done in the future.

DR. HARRISON BLACK, Boston, Mass.—I have enjoyed very much the presentations of Drs. Glover and Nichols, and their important contributions to this subject. On Dr. Harken's services in Boston we have wrestled with this difficult problem over the years. It is apparent that, although some patients have been greatly helped, the ultimate salvage when they are followed for a significant time leaves one dissatisfied with all the techniques currently available including intra-auricular baffles, annulus distortion, external annulus constriction, and external bolstering of the posterior herniation of the valve complex. At the risk of seeming repetitious, however, I believe I should re-emphasize several principles which constitute a distillate of our experience in dealing with this problem.

First of all, as we have been reminded by the authors, it is extremely important not to immobilize the *leaflets*. Any procedure which does this, as we have stressed on numerous occasions in the past, is going to result in damaging obstruction to forward flow. We cannot trade stenosis *for insufficiency*.

Second, the aneurysmal dilatation of the left auricle which occurs in the advanced stages of this disease *must* be corrected. Patients with predominant mitral insufficiency do not become *significantly* symptomatic, for the most part, until an aneurysmal *dilatation* of the left auricle has occurred and left ventricular failure has supervened. As the systolic jet weakens the *auricular* wall the chamber enlarges to enormous size and the pull of gravity in the erect position causes it *to* fall posteriorly *toward* the diaphragm. This in turn results in a sliding hernia of the valve leaflets at the posterior commissure which aggravates their incompetence by pulling them further apart. It seems reasonable to assume that this particular anatomic situation accounts for the concentration of most significant mitral insufficiency about the posterior commissure. Therefore, any technique for the correction of mitral insufficiency, in addition to reducing the circumference of the annulus and preserving leaflet mobility, must take into consideration this third dimensional concept by reducing the herniation and possibly also by plicating the redundant auricular wall.

Finally, in evaluating the results of any procedure for the correction of mitral insufficiency we must limit ourselves to pure cases. It is, of course, obvious that no one can choose to operate only on pure incompetence since many mixed cases are invariably encountered. In assessing results, however, it is well to recall that Ellis* in analyzing the outcome of valvuloplasty in the first 500 of our cases of predominant mitral stenosis, found on long-term follow-up that even when marked insufficiency was found at operation, the correction of the existing stenosis alone resulted in improvement in 50 per cent of the cases. When moderate degrees of incompetence were encountered, 70 per cent showed improvement after correction of the stenosis alone.

As concerns the evaluation of what one has accomplished immediately, the reduction or elimination of the palpable jet is of course *important provided it is observed at an* adequate blood pressure. Changes in murmurs are influenced by so many factors, including pericardial and pleural effusions in the early postoperative period, that they are often unreliable. The V wave in the pulmonary capillary or left auricular tracing has not correlated at all well, in our experience, with the degree of mitral insufficiency. We are currently investigating differential dye dilution curves during left and right heart catheterization and believe that these may be useful in quantitating regurgitation when correlated with cardiac output studies.

Our point of view concerning the surgical therapy of mitral insufficiency at this time thus differs from that expressed yesterday by Dr. Harken regarding aortic stenosis. We now feel that the majority of these cases are going to require the use of an open technique for constricting the mitral annulus and reducing the herniation of the leaflets.

DR. LEWIS H. BOSHER, Richmond, Va.—I should like to discuss Dr. Glover's paper. Certainly we owe a great deal to Dr. Glover and Dr. Davila, who have developed this procedure for mitral insufficiency. We have operated upon 5 patients with free mitral insufficiency by this technique. These were all advanced cases with severe cardiomegaly. There have been two deaths among the 5, both perhaps preventable deaths with further experience on our part. Certainly the placement of this purse-string suture is difficult, and I think we must all appreciate that a good deal of work in the dog laboratory and also in the post-mortem room is necessary before the procedure is used in the operating room.

In one of our patients, we encountered a pericardial symphysis which made the landmarks difficult to recognize. In this patient, as proved at post-mortem examination six days later, we found that the suture had been passed beneath one of the tricuspid leaflets through the tricuspid orifice and, as it had been tightened, apparently it had come into contact with the ventricular myocardium with subsequent cutting through of the suture as determined at post-mortem. We feel that even if the suture is placed accurately there may be a tendency for it to cut through.

(slide) I think this perhaps illustrates that point. This is a left atrial tracing made at the time of operation before the purse-string suture is tightened. You will notice the high V wave and the high atrial pressures. After the suture is tightened we have a very respectable left atrial tracing. However, on the fourth postoperative day this patient's systolic murmur returned and we felt that perhaps the suture had cut through. Four weeks later we did another left atrial puncture and it is evident that considerable mitral insufficiency has returned. It may be that in this patient there was considerable bunching of the pericardial tube which was used, and this may have accounted for that.

---

*Ellis, L. B., and Harken, D. E.: Factors Influencing the Late Results of Mitral Valvuloplasty for Mitral Stenosis, Ann. Int. Med. 43: 133-142, 1955.

We have one patient in whom there is certainly a very creditable result (slide). Here are the preoperative left atrial tracings, here are the tracings three months postoperatively. The mean pressure has dropped from 28 to 7 and the left ventricular pressure has dropped from 15 to 7. This patient has no systolic murmur, the precordium is very quiet, the patient is markedly improved clinically, and we are very happy with the result.

I think in some cases if considerable tightening of the purse string is necessary in order to eliminate the insufficiency, it may be well to use another technique to supplement this procedure; it may not in itself be entirely sufficient in all cases.

DR. JULIO C. DAVILA, Philadelphia, Pa.—I should simply like to [illegible] one comment, perhaps repeating what Dr. Bosher has said, that is to urge anyone who wishes to attempt this procedure to spend a lot more time than he thinks is necessary in the laboratory prior to attempting it in the patient. I think anyone who does [illegible] will find considerable difficulty in learning the technique at first, but as he acquires, shall we say, a spatial concept of the pertinent cardiac anatomy, he will find that the technique is quite easy, with proper orientation.

I should like to make two comments about the point which Dr. [illegible], which was a very good one, how to judge the degree of correction of regurgitation. We are not yet at the point where we can measure regurgitation with any degree of accuracy, although some efforts are being made to do so by indicator techniques and these show considerable promise. The jet, per se, is not a satisfactory indicator of how much regurgitation has been modified because the jet will change with changes in systemic pressure. The best evidence of the degree of correction obtained that we have found has been the measurement of left atrial, left ventricular, and aortic pressures so that we can gauge the degree of change in the left atrial pressure correlated with the other pressures mentioned.

I would like to emphasize again the technical point that this procedure requires extensive study of the pertinent anatomy. It should be done with precision and with absolute understanding of the anatomy of the heart.

DR. EARLE B. KAY, Cleveland, Ohio.—I should like to discuss Dr. Nichols' very interesting paper. I believe we were one of the first to stress the significance of the dilated mitral annulus and the separated posteromedial commissure in the pathology of mitral regurgitation. Over two years ago we developed a technique demonstrating various ways of reducing the size of the annulus at the posteromedial commissure in order to obliterate the regurgitant jet of blood. The particular variation subsequently [illegible] for obvious reasons mentioned by Dr. Nichols in closing, as illustrative of our technique was discarded [illegible] after only several months. Since the follow-up in the series has been for [illegible] of 8 patients who were operated upon two years ago employing this principle, I thought it might be of interest to the group to relate [illegible] about what one would expect. Those patients who had primarily "pure" mitral regurgitation in whom the valve cusps were fairly pliable so that the commissure could be [illegible] all have done well. Those in whom there was calcification and rigidity of the cusps [illegible], and where the posteromedial commissure could either not be approximated or brought together only under tension, those patients have done poorly and I believe [illegible] technique and not as precise as might be desired.

In any event, evaluation is difficult [illegible] obtained with the open direct vision technique employing a pump oxygenator. [illegible] developing that a better [illegible] following the same basic principles.

DR. RICHARD H. MEADE [illegible]—When I was in Dr. Crafoord's Clinic in Stockholm, Dr. Crafoord [illegible] Dr. Senning, who said, "I am going to try an open [illegible] mitral regurgitation. I am plan[illegible] a circumferential suture of the annulus." I said, "That is very interesting; I just came from a meeting of the American Association for Thoracic Surgery, where the operation was discussed by Dr. Glover." I wonder if Dr. Senning or Dr. Crafoord have done any more.

DR. H. HANSON, Stockholm, Sweden.—This discussion was not returned. EDITOR.

DR. CHARLES P. BAILEY, Philadelphia, Pa.—First, I want to point out that polar cross-fusion is entirely original with Dr. Nichols—I have had no part in its development except that of offering encouragement. I have had some little part in the handling of some of the cases reported by Dr. Nichols, and I want to emphasize what Dr. Glover has said about the desperate condition preoperatively in most of these individuals. One of the patients in this series was in a hospital under our care for nine months before she could be brought into condition for surgery. Dr. Nichols refuses to talk about the improvement as observed clinically, and yet our cardiologists uniformly are extremely enthusiastic and think the procedure is very good. I am tremendously impressed by the consistently great reduction in the regurgitant jet as recognized by intracardiac palpation at the time of operation. The improvement achieved at surgery transcends anything I have ever been able to accomplish with any other procedure for mitral regurgitation. Of course we must wait to find out what the long-term results will be. I do hope that either Dr. Davila's or Dr. Nichols' procedure will prove to be the final answer. If neither does, we will just have to keep on trying.

I have, as Dr. Glover and Dr. Davila well know, "viewed with alarm" the procedure which they have just reported because I recommended and carried out a somewhat similar operation three and one-half years ago in attempting to correct aortic insufficiency. Those of you who have inserted a Hufnagel valve know how difficult it is to put a circumferential suture accurately about the plastic ring that holds the valve against the aortic wall. This is done under direct vision and with the help of the flanges which are built into the ring. These mitral valve rings are about twice the diameter, are nearly the same thickness, and have no flanges. By a closed, digitally guided technique, it seems inconceivable that a constricting band can be placed accurately about the entire circumference of the mitral (or aortic) ring. Yet, if it slips off any portion of the annulus, it will lose all its constricting effect and, in some instances, will cut through into the lumen. I think my long-term failures with circumferential constriction for aortic insufficiency may be paralleled by similar failures with mitral constriction. Almost all those patients are now dead. The band seemed to slip off the annulus after weeks or months or years. Sometimes it cut through into the aortic or ventricular lumen; other times the regurgitation came back. Nevertheless, if only because they directed our attention toward the importance of the annulus in the correction of mitral insufficiency, we owe Drs. Davila and Glover a tremendous debt.

DR. ROBERT P. GLOVER (Closing).—I should like to thank all the discussants and say a few words in closing. I want to emphasize as strongly as possible the vicious cycle through which mitral insufficiency passes over the years. Any procedure that does not pretty well abolish the existing mitral insufficiency, or if for some reason an appreciable degree of insufficiency remains uncorrected, dilatation of the ring as the years go on will progressively increase. I believe that it is essential to permanently fix the *entire* ring by total constriction; otherwise annular dilatation will continue and the vicious cycle will proceed unabated.

Dr. Black spoke of the aneurysmal dilatation of the left atrium. These atria get as large as the largest of Florida grapefruit and we have been sorely tempted on a number of occasions, after tying the constriction purse-string suture and observing the tense and distended atrial wall to relax and even become wrinkled, to clamp off and resect much of the now redundant atrium. We have not done this as yet, however, so that no confusion would exist as to the proper postoperative evaluation of the purse-string method per se, but I think it entirely conceivable that some of these tremendous atria can be amputated and refashioned to a reasonable size. These atria no longer beat, they are incapable of contraction, so they might well be made as small as possible.

With regard to Dr. Bosher's statement regarding possible suture cut through into the atrium, such has not occurred when the suture was properly placed. I might state that in some tens of dogs followed well over a year, and we still have twenty to be followed for the next four or five years, there has been no cut through. In our oldest postoperative patient, one on whom an autopsy was performed at eight months, the purse string was intact, well fixed by fibrosis, and no evidence of cut through was present.

The application of circumferential suture to aortic insufficiency is, in my opinion, questionable. The anatomy, pathology, and the actual tissues themselves present in aortic valve disease would not seem to be suitable for a circumferential constriction of this type. The wall of the aortic ring, thin and bare, will not tolerate constant constriction and I believe cut through would be inevitable; but the tissues concerned in the mitral annulus, being muscular as they are, will tolerate constrictive pressures and actually incorporate the purse string within a fibrous capsule. Thus, the least of our problems is concerned with cut through; our greatest problem is the status of the patient's valve itself. Everyone in this audience who has digitally explored a heart valve knows that one cannot predict, prior to such exploration, the actual tissue status of a valve nor what can be accomplished surgically. One can only accomplish what the valve in its diseased state will allow. In 2 of the cases just reported there was actually no remaining functionable valve tissue. The leaflets were contracted right back into the myocardium so that the chordae tendineae actually emerged from a contracted and fixed ridge of scar at the annulus. Despite almost complete constriction of the purse string in these instances nothing but a reduction in size of the mitral orifice could be effected for there were no leaflets to coapt. This situation cannot be predicted clinically and remains for exploration to reveal.

I agree that perhaps total mitral annular purse string is not the final answer, but it is the only procedure of the many tested in our laboratory that routinely will correct regurgitation and it has done so to date, so far as the valves have allowed, in the clinical cases reported.

DR. HENRY T. NICHOLS (Closing).—I should like to thank all the discussants and, if I may, I should like to show a slide.

(slide) In Dr. Kay's discussion, he spoke of having done this same procedure two years before. I think there is a little variation and I have taken the liberty of making this slide from his article. It shows that he has attacked the same portions of the mitral annulus and I certainly give him the credit he deserves. However, I would like to point out that he has used buttons on the outside of the heart in this one of his procedures and that there is considerable soft tissue interposed between the two points of counterpressure, a button on one side and one on the other. I have doubt as to whether the two areas of the annulus will grow together. It might be that this is not important; I do not know. Also, we have worried about the possibility of leaving bare sutures inside the atrium in fear of developing infection.

(slide) Although perhaps this slide is erroneous, I thought he was not pulling the annulus together because of this diagram. I realize that a diagram can be misleading, but it is Kay's diagram and I did get the impression that he was merely distorting the annulus, as shown in these diagrams, before and after.

So far as the history of the attack upon the mitral annulus is concerned, I would like to say that Bernheim, back in 1909, was the first man to attack the mitral annulus, so far as I know. He did this in an attempt to produce mitral stenosis in dogs. [illegible] did plicate the annulus, and passed two parallel sutures, one starting heart, piercing the annulus in two places and then emerging from the front of the heart. The second suture was placed parallel to the first. He tied the two free ends on the back together and followed this by tying down the ends in front. Thus he pulled one part of the annulus against the other. In the same article, he described a circumferential suture that he applied. He called it a circular ligature around the mitral annulus, and he passed the needle underneath the coronaries to the left ventricle. I feel that if this is not the same as the Davila technique, it probably is very similar. However, I think Dr. Glover and I, and all of us, can take some comfort in the fact that Bernheim was not successful in producing mitral stenosis.

# PATENT DUCTUS ARTERIOSUS IN INFANCY

S. Richard Bauersfeld, M.D.* (by invitation), Paul C. Adkins, M.D.** (by invitation), and Edward M. Kent, M.D.***
Pittsburgh, Pa.

In the usual case of patent ductus arteriosus, there is little if any difficulty during infancy. If the diagnosis is made in early life, operation may be safely deferred until the age of 3 years or more. It is an established fact, however, that a large patent ductus arteriosus may produce severe strain on the circulation, which may ultimately lead to cardiac failure and death during infancy.[6, 7, 10] Consequently, early recognition of this condition is necessary in those infants who are showing evidence of cardiac embarrassment. The purpose of this paper is to discuss those cases of patent ductus arteriosus in which surgical intervention was felt to be indicated before the age of 24 months.

The time of normal anatomic closure of the ductus arteriosus has been discussed in many reviews. Although anatomic patency may persist up to six weeks after birth, most writers feel that functional closure occurs within the first two weeks of life. The ductus which has not closed within the first two months of life will probably remain patent, and the possibility of delayed closure during childhood is remote. The continuous murmur, characteristic of a patent ductus arteriosus, is due to the flow of blood through the ductus during systole and diastole. Since, at birth, there is little difference in pressure between the systemic and pulmonary circulation, there is little flow from the aorta to the pulmonary artery. Consequently, only a systolic murmur is present. With development of the infant, the pressure rises in the systemic circulation and falls in the pulmonary circulation. Eventually, there is a considerable difference in pressure between the aorta and pulmonary artery, resulting in flow of blood through the ductus from the aorta to pulmonary artery during both systole and diastole. At this time, a continuous murmur will be present. Characteristically, these pressure changes occur during the first six months of life, so that prior to the age of 6 months, a systolic murmur only may be present. In some instances, the systolic murmur may not appear until later and, occasionally, may never appear when there is marked pulmonary hypertension.

---

From the Department of Cardiology, Children's Hospital of Pittsburgh, and the Division of Thoracic Surgery, University of Pittsburgh School of Medicine, Pittsburgh, Pa.

This study was aided by grants from the Western Pennsylvania Heart Association and the Kiwanis Club of Pittsburgh.

Read at the Thirty-sixth Annual Meeting of The American Association for Thoracic Surgery at Miami Beach, Fla., May 7 to 9, 1956.

*Associate Professor of Pediatrics, University of Pittsburgh School of Medicine, and Director of Cardiac Clinic, Children's Hospital of Pittsburgh.

**Instructor in Surgery, George Washington University School of Medicine, Washington, D. C.

***Associate Professor of Surgery and Chief of Thoracic Surgical Section, University of Pittsburgh School of Medicine.

The findings in a typical case of patent ductus are well known. The most outstanding diagnostic finding is the presence of a continuous murmur at the upper left sternal border. Other salient features are: accentuation of the pulmonary second sound, absence of cyanosis, a wide systemic pulse pressure and fluoroscopic or roentgenographic evidence of increased pulmonary blood flow. In addition, there may be varying degrees of enlargement of the left atrium and left ventricle. Under certain conditions, as previously noted, patency of the ductus arteriosus does not produce a continuous murmur. These patients with only a systolic murmur have been given the term "atypical patent ductus." The term "atypical" is not accurate, however, since the absence of a continuous murmur may be typical for the specific situation, as in young infants. It is not within the scope of this paper to discuss the other causes of a patent ductus arteriosus without a continuous murmur.

Over the past three years, a diagnosis of patent ductus arteriosus was made and confirmed by operation or autopsy on 74 children of all ages at the Children's Hospital of Pittsburgh (Table I). This series does not include any case in which a diagnosis has been made but not yet confirmed by direct visualization. No case was included where a patent ductus arteriosus was present as a compensatory lesion, as in cyantoic heart disease. There are, however, in this series, those cases with an additional anomaly of the heart or great vessels, but in which the patent ductus arteriosus was not considered a compensatory lesion. There were 55 females and 19 males in this series. This corresponds with other reported series, in that there is a preponderance of females. Of the entire group of 74 cases, 22 (30 per cent) were under the age of 2 years. This paper is primarily an analysis of these 22 infants, since it was in this group that the diagnosis was least frequently obvious and, conversely, the same group which presented most often with symptoms which could be attributed to the patent ductus arteriosus. Of the 74 cases of all ages, there were 16 (22 per cent) in whom a systolic murmur only was heard prior to operation. As may be seen from Table I, 12 of the 16 patients in whom a continuous murmur was absent were under the age of 2 years.

TABLE I. AGE, SEX, AND TYPE OF MURMUR IN 74 CASES OF PATENT DUCTUS ARTERIOSUS

| AGE GROUP | NUMBER OF CASES | SEX |  | MURMUR |  |
|---|---|---|---|---|---|
|  |  | MALE | FEMALE | SYSTOLIC | CONTINUOUS |
| Under 24 months | 22 | 9 | 13 | 13* | 9 |
| Over 24 months | 52 | 10 | 42 | 4 | 48 |
| Total | 74 | 19 | 55 | 17* | 57 |

*One had systolic and diastolic murmer.

Analysis of the symptoms and physical findings in the children under the age of 24 months (see Table III) shows that nearly all of these children had a history of poor weight gain since birth. Also, many had dyspnea or frequent respiratory infections and 5 gave a history of intermittent minimal cyanosis, usually accompanying respiratory infections. Based on the Boston Children's Medical Center Anthropometric Charts, all but one of these infants were in the third percentile or less for their age and weight. Their birth weights in general were slightly below average and only 4 were under five pounds at birth.

TABLE II WEIGHT CHANGES IN 22 CASES OF PATENT DUCTUS ARTERIOSUS UNDER 24 MONTHS OF AGE

| | PREOPERATIVE | | | | POSTOPERATIVE | | |
|---|---|---|---|---|---|---|---|
| CASE | AGE (MONTHS) | BIRTH WEIGHT (POUNDS) | WEIGHT AT OPERATION (POUNDS) | PERCENTILE | LENGTH OF FOLLOW UP (MONTHS) | PRESENT WEIGHT (POUNDS) | PERCENTILE* |
| 1 S M | 5½ | 6 | 9½ | <3 | 15 | 18 | <3 |
| 2 N A | 7 | 6¾ | 11½ | <3 | 1 | 18 | 10th |
| 3 D D | 10 | 6¼ | 11 | <3 | 7 | 18 | <3 |
| 4 T O | 4½ | 7¼ | 9¾ | <3 | 26 | 25 | 3rd |
| 5 J N | 16 | 4¾ | 10½ | <3 | 12 | 28 | 10th |
| 6 L W | 8 | 5½ | 10 | <3 | 23 | 22 | <3rd |
| 7 L H | 23 | 5 | 18 | <3 | 11 | 25 | <3rd |
| 8 R M | 14 | 3¾ | 18 | 3rd | 16 | 23½ | <3 |
| 9 M B | 8 | 4¾ | 9 | <3 | Coarctation and PDA, Died 3 mos postoperative | | |
| 10 G G | 4 | 8¾ | 12 | <3 | PDA and IASD, Died 3 mos postoperative | | |
| 11 M S | 2 | 6 | 6½ | <3 | 7 | 10½ | <3 |
| 12 P K | 6 | 8 | 11 | <3 | No operation | | |
| 13 T S | 20 | 9½ | 16½ | <3 | 27 | 39 | >75<90 |
| 14 S B | 6 | 5 | 10¼ | <3 | 3 | 16 | 10th |
| 15 A F | 8 | 5¾ | 15 | 3rd | 18 | 22 | 3rd |
| 16 C H | 21 | 3½ | 16 | <3 | 11 | 20½ | <3 |
| 17 R C | 7 | 5¼ | 9 | <3 | 10 | 21½ | >10<25 |
| 18 N M | 12 | 8½ | 18 | 25 | 1 | | |
| 19 K B | 3 | 6¾ | 6½ | <3 | No operation | | |
| 20 M W | 2 | 6 | 7½ | <3 | 1 | 9½ | <3 |
| 21 F Y | 6 | 4¾ | 8¾ | <3 | 1 | | |
| 22 D N | 5 | 5¾ | 10½ | <3 | 1 | | |

*Based on The Boston Children's Medical Center Anthropometric Charts

Table III lists the physical findings in this group of 22 infants. In 9 (41 per cent) of these infants, a continuous murmur was heard. Of the remaining 13 infants, 12 had only a systolic murmur located along the left sternal border. In one infant, the murmur was systolic and diastolic but not continuous. In most of this group *the thrill and murmur were of maximal intensity* in the pulmonic area. However, in 3 infants (Cases 2, 9, 10) with proved patent ductus arteriosus, the thrill and murmur were maximal along the lower left sternal border. All showed roentgenographic evidence of cardiac enlargement and increased pulmonary vascular markings. In those infants where blood pressure was obtained by the flush technique, the systolic pressure only is listed.

The electrocardiogram showed considerable variation (Table IV). In 16 cases the axis was normal, 11 of these had no hypertrophy from the precordial leads, in 3 there was left ventricular hypertrophy and in 2, right ventricular hypertrophy. Right axis deviation plus right ventricular hypertrophy was present in 5 infants. In one infant, there was left axis deviation and no evidence of ventricular hypertrophy from the precordial leads. Four of the 7 cases with right ventricular hypertrophy had additional cardiac anomalies, one with coarctation, 2 with interventricular septal defects and one with reversed flow through the ductus. There was no evidence of additional cardiac anomalies in the remaining 3 cases. One of this group died and, at autopsy, the only abnormality found was a large patent ductus arteriosus.

TABLE III. SIGNS AND SYMPTOMS IN 22 PATIENTS UNDER 24 MONTHS OF AGE WITH PATENT DUCTUS ARTERIOSUS

|  | HISTORY |  |  |  | PHYSICAL FINDINGS |  |  |  |
|---|---|---|---|---|---|---|---|---|
|  |  |  |  |  |  | MURMUR |  |  |
| CASE | POOR WEIGHT GAIN | RESPIRATORY DIFFICULTY | INTERMITTENT CYANOSIS | THRILL | PULMONARY SECOND SOUND | CONTINUOUS | SYSTOLIC | BLOOD PRESSURE |
| 1. S. M. | + | + | + | 0 | Inc | — | 2 and 4 LICS | — |
| 2. N. A. | + | + | 0 | + | Inc | — | 4th LICS | 120/80 |
| 3. D. D. | + | 0 | 0 | + | Inc | — | 3 and 4 LICS | — |
| 4. T. O. | + | 0 | 0 | 0 | Inc | 2 and 3 LICS | — | 100 |
| 5. J. N. | + | 0 | + | + | Inc | — | 2nd LICS | 86 |
| 6. L. W. | + | + | 0 | 0 | Inc | — | 2 and 3 LICS | 94 |
| 7. L. H. | + | 0 | 0 | + | Inc | — | LSB | 130/80 |
| 8. R. M. | + | 0 | + | 0 | Inc | — | LSB | 70 |
| 9. M. B. | + | 0 | 0 | 0 | Inc | — | 4th LICS | Arm 115 Leg 50 |
| 10. G. G. | + | + | + | 0 | Inc | — | 4 and 5 LICS | 115/65 |
| 11. M. S. | + | + | 0 | 0 | Inc |  | LSB | — |
| 12. P. K. | + | + | + | + | Inc | 3rd LICS | — | 75 |
| 13. J. S. | + | 0 | 0 | + | Inc | 2nd LICS | — | 120/60 |
| 14. S. B. | + | 0 | 0 | 0 | Inc | 2nd LICS | — | — |
| 15. A. F. | 0 | 0 | 0 | + | Inc | 2 and 3 LICS | — | 100/50 |
| 16. C. H. | + | + | 0 | 0 | Inc | 2 and 3 LICS | — | — |
| 17. R. C. | + | + | 0 | + | Inc | 2 and 3 LICS | — | 100 |
| 18. N. M. | 0 | 0 | 0 | + | Inc | 2 and 3 LICS | — | — |
| 19. K. B. | + | + | 0 | 0 | Inc | — | LSB |  |
| 20. M. W.* | + | 0 | 0 | + | — |  |  | 120/60 |
| 21. F. Y. | + | 0 | 0 | + | Inc | 2nd LICS | — | 140 |
| 22. D. N. | + | + | 0 | 0 | — | — | 3 and 4 LICS | 120 |
| Total | 20 | 10 | 5 | 11 | 20 | 9 | 12* | — |

*Systolic and diastolic but not continuous at 4 LICS.

Key: Inc—increased.
LICS—left intercostal space.
LSB—left sternal border.

TABLE IV. SPECIAL STUDIES IN 22 CASES OF PATENT DUCTUS ARTERIOSUS UNDER 24 MONTHS OF AGE

|  | X-RAY AND FLUOROSCOPY |  |  |  |  |  | ELECTROCARDIOGRAM |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|
| CASE | C-T RATIO | LV | LA | RV | RA | PULMONARY VASCULARITY | AXIS | PRECORDIAL LEADS | AORTOGRAM | FINAL DIAGNOSIS |
| 1. S. M. | .60 | + | 0 | 0 | 0 | Inc | BAL | LVH | PDA | PDA |
| 2. N. A. | .63 | + | ± | 0 | 0 | Inc | BAL | — | PDA | PDA |
| 3. D. D. | .58 | ++ | + | 0 | 0 | Inc | BAL | LVH | PDA | PDA |
| 4. T. O. | .55 | ++ | + | 0 | 0 | Inc | BAL | — | PDA | PDA |
| 5. J. N. | .62 | ± | 0 | ++ | ± | Inc | RAD | RVH | PDA | PDA |
| 6. L. W. | .62 | ++ | +++ | 0 | + | Inc | BAL | RVH | Unsatis. | PDA and IVSD |
| 7. L. H. | .58 | + | 0 | 0 | 0 | Inc | BAL | — | — | PDA |
| 8. R. M. | .56 | + | 0 | + | ± | Inc | RAD | RVH | — | PDA |
| 9. M. B. | .58 | +++ | 0 | + | 0 | Inc | RAD | RVH | PDA and Coarc. | PDA and Coarc. |
| 10. G. G. | .63 | + | 0 | 0 | 0 | Inc | LAD | — | PDA | PDA and IASD |
| 11. M. S. | .60 | ± | 0 | +++ | 0 | Inc | RAD | RVH-LVH | PDA | PDA and IVSD |
| 12. P. K. |  | + | 0 | + | + | Inc | RAD | RVH | — | PDA |
| 13. J. S. | .58 | ± | 0 | 0 | 0 | Inc | BAL | — | — | PDA |
| 14. S. B. | .60 | ± | 0 | + | 0 | Inc | BAL | RVH | — | PDA |
| 15. A. F. | .60 | ++ | ++ | 0 | 0 | Inc | BAL | — | — | PDA |
| 16. C. H. | .53 | + | ± | ++ | ++ | Inc | BAL | — | — | PDA |
| 17. R. C. | .6– | ++ | ++ | 0 | 0 | Inc | BAL | — | — | PDA |
| 18. N. M. | .54 | ++ | ++ | 0 | 0 | Inc | BAL | — | — | PDA |
| 19. K. B. | .63 | ++ | ++ | ± | ++ | Inc | BAL | — | — | PDA |
| 20. M. W. | .50 | + | + | + | 0 | Sl. Inc | BAL | LVH | PDA | PDA |
| 21. F. Y. |  | 0 | ± | 0 | 0 | Sl. Inc | BAL | — | — | PDA |
| 22. D. N. | .50 | + | + | 0 | 0 | Inc | BAL | — | PDA | PDA |

Key: C-T Ratio—Cardio-thoracic ratio.
LVH—Left ventricular hypertrophy.
RVH—Right ventricular hypertrophy.
BAL—Balance.
LAD—Left axis deviation.
RAD—Right axis deviation.

On the basis of a continuous murmur in the pulmonic area and other substantiating evidence by physical examination and fluoroscopy of the heart and lungs, it was possible to make a diagnosis of patent ductus arteriosus in 9 of these infants when first seen. In the remaining 13 infants, the physical findings were suggestive of a patent ductus arteriosus; in these cases, however, the absence of a continuous murmur made it impossible to differentiate this from other lesions causing a left-to-right shunt. The lesion we considered most difficult to differentiate from a patent ductus arteriosus in these infants was a high ventricular septal defect. Both a patent ductus arteriosus and a high ventricular septal defect may present with a similar history of poor weight gain, frequent respiratory difficulty, and a heart murmur heard during early life. Both conditions may present a systolic thrill and murmur heard along the left sternal border and a loud pulmonary second sound. The location of the thrill and murmur may not be helpful in differentiating between a patent ductus and a ventricular septal defect in these infants, since in several of our cases with proved patent ductus arteriosus, the thrill and systolic murmur were heard at the lower left

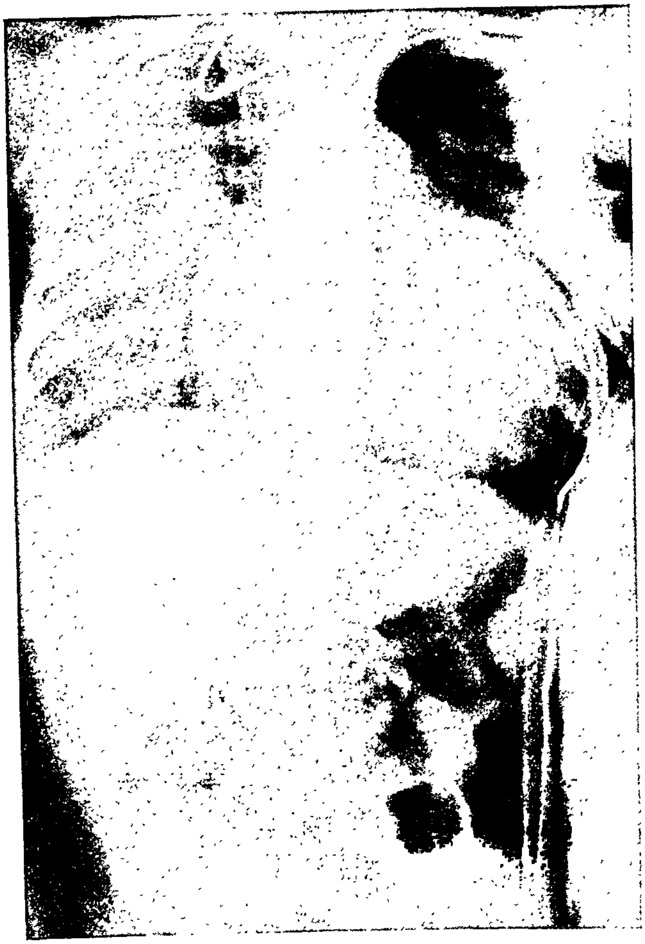


Fig. 1A.—Normal aortogram.

sternal border (Cases 2, 9, 10). Both conditions may exhibit increase in the over-all heart size and fluoroscopic evidence of increased pulmonary blood flow.

Consequently, in those infants seen with the above findings, who showed evidence of poor development and cardiac enlargement, it was felt that additional studies were indicated if a patent ductus arteriosus could not be ruled out as the cause of the left-to-right shunt. Since the infants in this group were quite small, the average weight being eleven pounds, retrograde aortography was considered more practical than cardiac catheterization to establish the presence or absence of a patent ductus arteriosus. One infant (Case 8) had findings suggestive of a patent ductus arteriosus with reversal of flow. This infant had rather striking intermittent cyanosis which was more marked in the lower extremities. In this case the diagnosis was confirmed by angiocardiogram. The diagnosis of patent ductus arteriosus was established by retrograde aortogram in 9 infants.

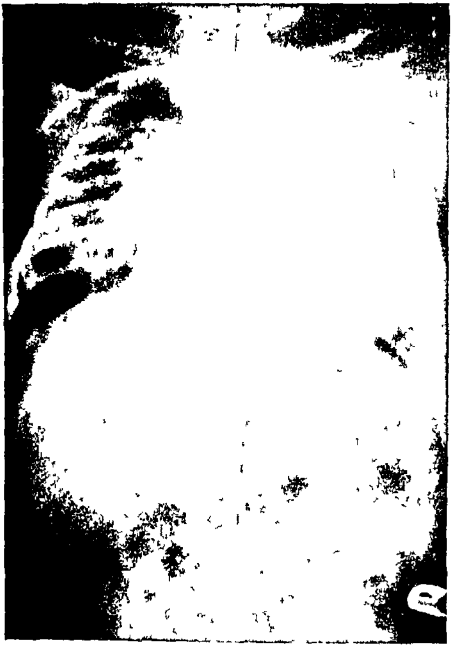


Fig. 1B.—Aortogram showing simultaneous filling of the aorta and pulmonary artery.

In the majority of cases, retrograde aortography was carried out under general endotracheal anesthesia and a modification of the method described by Keith was used. The carotid sheath was exposed through a 5 cm. trans-incision at the anterior border of the left sternocleidomastoid muscle. The left common carotid artery was freed up and two heavy silk ligatures were passed around it but not tied. Using these ligatures to control bleeding, a 3 mm. longitudinal opening was made in the carotid artery. A No. 240 polyethylene catheter was then passed proximally in the carotid artery for a distance of 2 to 3 cm. The lower ligature was then tied once around the vessel to hold the catheter in place. It was found by trial and error that a better concentration of the contrast medium was obtained if the tip of the catheter lay somewhat above the junction of the aorta and the left common carotid artery. After giving a test dose to determine sensitivity, a rapid injection of 6 to 8 ml. of 35 per cent Diodrast was made into the carotid artery through the cannula, and a single anteroposterior chest x-ray taken at the exact moment of completion of the injection. During injection of the dye, the opposite common carotid artery was compressed by the anesthesiologist. After the film had been taken, the catheter was withdrawn and the opening in the vessel closed with a continuous suture of 6-0 arterial silk. The remainder of the incision required only two or three fine silk sutures for closure. Simultaneous demonstration of the dye in the aorta and pulmonary arteries by x-ray was considered diagnostic of an aorticopulmonary shunt (Fig. 1.).

Using this technique, retrograde aortography was performed on 43 infants during this three-year period to determine the presence or absence of a patent ductus arteriosus. There were two deaths which could be ascribed to aortography, both early in the series, an over-all mortality of 4.6 per cent. It was felt that one of these deaths was definitely associated with an overdose of the contrast medium. The other infant demonstrated an interventricular septal defect, coarctation of the aorta, and subendocardial fibroelastosis at autopsy. If a patent ductus arteriosus was demonstrated by aortography, surgical closure was usually carried out within a week.

In one infant, the aortogram was technically unsatisfactory, but a patent ductus arteriosus was demonstrated at operation. In one case, a coarctation of the aorta and a patent ductus were suspected before aortography and this diagnosis was confirmed. In another, a questionable coarctation was seen on the aortogram, but there was no evidence of narrowing of the aorta at operation.

Of the 22 infants with patent ductus arteriosus, 2 died before any definitive procedure could be carried out. One infant (Case 12) was admitted with pneumonia and found to have the physical findings of a patent ductus arteriosus. Despite intensive efforts, the infection could not be controlled to a point where the patient was considered a suitable candidate for anesthesia and the child died before operation could be carried out. Autopsy revealed only a large patent ductus arteriosus. Another infant (Case 19) was admitted with a severe respiratory infection. Although a continuous murmur was not audible, the physical findings were suggestive of a patent ductus arteriosus. Retrograde aortogram was contemplated, but before the respiratory infection could be controlled, the infant died suddenly. At autopsy, an uncomplicated patent ductus arteriosus was demonstrated.

Operation was carried out on the remaining 20 infants in this series. Those patent ducti which were 10 mm. or more in width were divided between clamps and the ends closed by continuous suture. The patent ducti smaller than 10 mm. in width were closed by the multiple ligature technique.[12] There were no operative deaths. In the infant (Case 8) with reversal of flow through the patent ductus, temporary occlusion of the ductus at operation produced no deleterious effect and division was carried out without difficulty. This patient has continued to progress satisfactorily.

In 3 infants, there was a residual thrill over the base of the heart after occlusion of the patent ductus. One of these infants (Case 10) died three months postoperatively and was found to have a large residual interatrial septal defect. The other 2 (Cases 6 and 11) are living and well and both are considered to have a residual interventricular septal defect. The one infant (Case 9) who had a coarctation of the aorta and an associated patent ductus arteriosus proximal to the coarctation, died three months postoperatively. In this case, resection of the coarctation, as well as division of the patent ductus, had been contemplated prior to operation. However, after divisions of the patent ductus, the infant's condition became so precarious that further surgery was precluded and the chest was closed. Therefore, since only the patent ductus arteriosus was divided in this infant, the case is included in this series.

Postoperative follow up on the remaining 18 infants, over a period from one to twenty seven months, reveals that all have done well. Nearly all have gained weight satisfactorily, have been less prone to respiratory infections, and have been less irritable.

## SUMMARY

Persistent patency of the ductus arteriosus is most frequently asymptomatic during infancy and early childhood. However, in some infants, a patent ductus arteriosus may cause failure to gain weight, respiratory infections, and cardiac enlargement. Many of these infants with symptoms referable to the patent ductus do not have a continuous murmur. Consequently, the correct diagnosis is not obvious. The physical findings in this group are similar to the findings in infants with left to right shunts at other levels. In these infants, when evidence of cardiac enlargement is present, the diagnosis must be established by special studies. During the period that this series was seen, operative procedures for closure of septal defects were not available to us. Consequently, retrograde aortography was considered the simplest method of establishing the presence or absence of a patent ductus arteriosus in small infants. With the successful development of techniques for open cardiotomy, cardiac catheterization is indicated to establish the exact level of the left to right shunt.

It is also felt that if an infant has a left to right shunt at two levels, such as an interventricular septal defect and a patent ductus arteriosus, these have an additive effect. Consequently, definite benefit may be obtained by closure of the patent ductus arteriosus.

The frequency of patent ductus arteriosus without a continuous murmur is somewhat higher in this group than in other reported series.[8] The majority of cases presenting with systolic murmurs alone were infants. Consequently, it is felt that a diagnosis of patent ductus arteriosus should be considered in noncyanotic infants presenting with a systolic murmur and a history of poor weight gain. If there is evidence of increased pulmonary vascularity and cardiac enlargement, the diagnosis may be established by retrograde aortogram. If a patent ductus arteriosus is demonstrated, surgical closure can be performed an a normal cardiovascular status restored.

## REFERENCES

1. Gross, R. E., and Hubbard, J. P.: Surgical Ligation of a Patent Ductus Arteriosus; Report of First Successful Case, J. A. M. A. 112: 729-731, 1939.
2. Gibson, G. A., quoted by Gould, S. E.: Pathology of the Heart, Springfield, 1953, Charles C Thomas, publisher, p. 43.
3. Gilchrist, A. R.: Patent Ductus Arteriosus and Its Surgical Treatment, Brit. Heart J. 7: 1-36, 1945.
4. Wells, H. G.: Persistent Patency of the Ductus Arteriosus, Am. J. Med. Sc. 136: 381-400, 1908.
5. Gould, S. E.: Pathology of the Heart, Springfield, 1953, Charles C Thomas, publisher.
6. Ziegler, R. F.: Atypical Patent Ductus Arteriosus, Proc. Internat. Symposium on Cardiovascular Surgery, Detroit, Mich., 1955, W. B. Saunders Company, pp. 129-134.
7. Adams, P., Jr., Adams, F. H., Varco, R. L., Dammann, J. F., Jr., and Muller, W. H.: Diagnosis and Treatment of Patent Ductus Arteriosus in Infancy, Pediatrics 12: 664-673, 1953.
8. Ash, R., and Fischer, D.: Manifestations and Results of Treatment of Patent Ductus Arteriosus in Infancy and Childhood, Pediatrics 16: 695, 1955.
9. Zeigler, R. F.: The Importance of Patent Ductus Arteriosus in Infants, Am. Heart J. 43: 553, 1952.
10. Dammann, J. F., Jr., and Sell, C. G. R.: Patent Ductus Arteriosus in the Absence of a Continuous Murmur, Circulation 6: 110, 1952.
11. Keith, J. D., and Forsyth, C.: Aortography in Infants, Circulation 2: 907, 1950.
12. Scott, H. W., Jr.: Closure of Patent Ductus Arteriosus by Suture-Ligation Technique, Surg., Gynec. & Obst. 90: 91, 1950.

## DISCUSSION

DR. FRANCIS DAMMANN, JR., Charlottesville, Va.—I certainly enjoyed this paper, as I have the entire meeting. I am very glad that I was privileged to come.

There are a few points I would like to re-emphasize and add in addition to what Dr. Adkins has said. It is very true that patent ductus in early infancy can cause death. It is true that unless one has a high level of suspicion, in many instances, the diagnosis will not be made or even thought of. Once it has been suspected I agree with the authors that the best and easiest technique for establishing the diagnosis is retrograde aortography. We have injected a 35 per cent solution of Diodrast into the left, not the right brachial artery, feeling that perhaps this keeps it away from the brain and may cut down the mortality. Obviously, this procedure does not rule out an aorticopulmonary shunt which may be missed unless the right brachial artery is used. I think we have established the diagnosis of patent ductus in about one half of the patients who have had aortograms. I would consider this an acceptable percentage because the frequency of cardiac decompensation and death in this group is high.

There is another aspect that I think should be mentioned. We have seen several infants who have come out of cardiac failure and then developed a right-sided shunt. This indicates progressive pulmonary vascular disease. It seems to me that, if this process is to be avoided, early intervention, early interruption of the high pressure in the lungs, is very definitely indicated.

MR. RAYMOND HURT, London, England.—I should like to report 26 cases of patent ductus arteriosus in infancy that have been operated upon by Dr. Frank Gerbode in San Francisco. These infants have all been admitted to hospital suffering from recurrent attacks of heart failure associated with respiratory infection. Almost all these patients had only a systolic murmur over the precordium, together with clinical evidence of heart failure. The diagnosis has been confirmed by retrograde aortograms done through the left brachial artery, by making a small incision under local anesthesia and introducing a needle into the artery under direct vision. Of the total number of infants studied approximately one fourth have had a surgically correctible lesion. All these ducti have been divided and there has been no mortality. The benefit from the closure has been most satisfactory, even in those cases that have had an associated abnormality.

DR. EGBERT FELL, Chicago, Ill.—I wish to re-emphasize the importance of the paper that was so well presented by Dr. Adkins. We have learned the hard way regarding these little infants, and to date we have had 18 infants, one year of age or less, with large open ducti. The first 2 were not recognized and came to post-mortem, 14 are living normally following closure of the ducti, the seventeenth was found to have an aortic septal defect which was successfully closed, and the eighteenth died three hours after surgery in heart failure believed due to overhydration.

We do not believe aortography is indicated, and believe that its use carries an unnecessary risk of major importance. Dr. Benjamin M. Gasul, our pediatric cardiologist, believes and clearly demonstrates that this anomaly can be diagnosed clinically in almost every patient. The infant is sedated so that a careful satisfactory physical examination, electrocardiographic and x-ray studies can be done. The machinery murmur is usually heard and a wide pulse pressure found. The heart is enlarged and pulmonary vascular markings are increased. If we carry out a thorough examination with a cooperative sedated infant, more successful diagnoses and cures of the so-called "malignant ductus" will be reported.

DR H. HANSON, Stockholm, Sweden.—This discussion was not returned. EDITOR.

DR. F. MASON SONES, JR., Cleveland, Ohio.—Over a six-year period, we have encountered 21 infants younger than one year of age, who proved to have patent ducti and who did not have continuous murmurs. I feel very strongly that the diagnosis in this situation is best made by cardiac catheterization. All these infants were studied by careful auscultation under excellent conditions, and in none of them were we able to recognize a continuous murmur. We were unable to tell the difference between a huge patent ductus and a large interventricular septal defect. In 20 of the 21 infants in whom the diagnosis was proved, it was possible actually to catheterize the ductus. This can be done safely and is not difficult from a technical standpoint. It seems to me that the major factor in favor of retrograde aortography is that it can be accomplished quickly and with little loss of time. Catheterization certainly is more painstaking and does take a great deal more time, but offers a greater margin of safety to the patient than is possible with retrograde aortography.

DR. HERBERT E. WARDEN, Minneapolis, Minn.—I rise to compliment Dr. Bauersfeld and his coauthors for their very fine paper. In addition, I would like to thank them for stressing the importance of the patent ductus arteriosus in the infant age group, and to reaffirm their feeling that the surgical closure of a patent ductus in the presence of an associated interventricular defect, as it often is, nets a marked clinical improvement in the patient. This is often true even if nothing is done for the septal defect.

The patent ductus arteriosus has been the most frequent associated extracardiac congenital anomaly encountered in the group of patients that we have seen with septal defects. To date all but one of these occurred in patients in this young age group. Preferentially, we feel that the ductus should be closed at the same time that corrective surgical repair of the ventricular septal defect is carried out. However, if this is not feasible and the patient's condition is deteriorating, a remission or at least a retardation or a slowing of the downhill course can be accomplished by the closure of the ductus even in the presence of a persistent ventricular shunt. We have seen this occur in one patient who was moribund. The presence of an interventricular defect was not suspected in him prior to thoracotomy and therefore suitable preparations for intracardiac repair were not available. However, the ductus was transected and the patient made a remarkable recovery following this closure and some months later successfully endured the surgical repair of the interventricular septal defect.

DR. H. BRODIE STEPHENS, San Francisco, Calif.—I enjoyed this presentation and believe it would be advisable, when we discuss this subject of patent ductus arteriosus in infants, that measurements be made of the diameter of the ductus by calipers and, in addition, measurement of the diameter of the aorta and pulmonary artery be carried out at the same time. I think by so doing and by documenting and reporting these measurements, we might obtain additional information on this interesting subject.

DR. S. RICHARD BAUERSFELD (Closing).—I certainly appreciate all the discussion of this paper which Dr. Adkins presented. I should like to make one or two comments about some of the statements that have been made.

First of all, we agree with Dr. Dammann about the importance of being aware of an atypical ductus. The diagnosis becomes easier when one realizes that a ductus can cause great difficulty and he is willing to hunt for this lesion. If one does not take all this trouble, one will not find it. We are indebted to Dr. Dammann for the initial studies of this condition. He reported a series of ducti very similar to our group in *Circulation* in 1952 or 1953. In our own group of patients the majority were less than one year of age, only 5 being over twelve months.

We agree with Dr. Fell that the pulse pressure is very important, although we have seen very large pulse pressures in patients with large ventricular defects. I certainly cannot agree with him about the continuous murmur. Likewise, in a recent report in *Pediatrics* from the Children's Hospital in Philadelphia, a large series of ducti were reported in which continuous murmurs were found in the majority of the patients. It has been our policy to go into the operating room and listen to these patients while they are under anesthesia, yet we have not been able to find continuous murmurs even though we know that a ductus is present. In this regard we agree with Dr. Sones that aortograms are necessary to demonstrate the ductus.

So far as measurements of the ductus are concerned, unfortunately we have not done this. We have approximated measurements. As a rule the ducti are as large as the aorta and in several cases the ductus was actually larger than the aorta. The statement about the risk of aortograms—this is true. It does carry a risk and in our own series this comes to some 5 per cent. On the other hand, the risk of missing the diagnosis of a ductus, and having the patient die, is even greater. Therefore, balancing these things, we certainly feel that aortography should be done. One thing that has struck us is that associated anomalies in these children are usually higher than are reported in other series of ducti. In our over-all group of some 74 patients, the incidence of additional defects amounted to 10 per cent. In this particular group it is almost 20 per cent.

At one time we thought the finding of right ventricular hypertrophy would help differentiate a patient with a patent ductus from one with an interventricular septal defect. However, as pointed out, 3 of our patients had right ventricular hypertrophy and nothing but a large ductus. At the present time, I do not believe that I can separate a patient with a ventricular defect from one with a ductus without going ahead with additional studies such as aortography.

# Letters to the Editor

Recently, the Editor received a letter from Dr Seymour Schotz of The Presbyterian Hospital in Philadelphia objecting to the discussion made by Dr Jerome R Head, of Chicago, of the papers dealing with anesthesia that were published in the October issue of the JOURNAL Dr Head's remarks were as follows

'The presentation of these interesting and excellent papers before this Association seems to me important and appropriate However much the anesthesiologists have tried to argue us out of the contention, the responsibility for the anesthesia lies with the surgeon, and for the surgeon to pass it entirely to the anesthesiologist is against Hippocratic principles and against the principles of modern law The findings presented in these papers also seem encouraging It means that what we had almost come to accept as a fated and unavoidable accident is becoming an understandable phenomenon and a preventable phenomenon Among all primitive peoples, when anything is not understood and is potentially disastrous, it is attributed to evil spirits, and I would confess that my attitude toward cardiac arrest has been heretofore largely on that basis One of the evil spirits has been the anesthesiologist, but more specifically and less facetiously, for a long time I placed my faith in ether As a young man I went into the operating room and was given a can of ether and a mask and told to put people to sleep They all lived, which was very surprising to me, and I attributed it entirely to the fact that you could not kill people with ether But I found out, over a period of years, that I had had three cardiac arrests under pure ether anesthesia all of them during induction I am sure now that this was because of inadequate relaxation and of multiple attempts to insert an intratracheal tube I was finally convinced that a long difficult induction with multiple attempts to insert a tube was bad I should have said also that one of my evil spirits was curare, I turned to Pentothal and nitrous oxide and to controlled respiration, and I put my faith in that, as one would put his faith in a god or a dance or whatever you have I got along very well with that until I realized that controlled respiration was of two kinds, either manual or mechanical Recently I had a case of cardiac arrest due, I feel sure, to the fact that the anesthesiologist became so interested in watching the resident put a catheter into a vein in the leg that he forgot to pump the bag That is a human error and, as a result of it, I have gone to mechanical respiration and to hyperventilation Hyperventilation may have certain drawbacks but they are much less than those of underventilation Therefore, at the present time as soon as I make the incision I begin to say—'The blood is black, come on, get going, the blood is black' I do that at intervals all through the operation and so far it has worked I have not had a cardiac arrest for a year

'These papers are very interesting and very important but they do not show what happens during cardiac arrest I feel that every anesthetic agent at the present time should be considered experimental, that any anesthesiologist who is not checking himself and being checked by blood oxygen readings, by $CO_2$ analyses and by the running electrocardiogram, is not being a good anesthesiologist The only way in which we will learn exactly what happens, exactly what causes cardiac arrest, is to have such data on a group of cases that have suffered cardiac arrest'

The letter from Dr. Schotz is as follows:

To the Editor of THE JOURNAL OF THORACIC SURGERY:

It is difficult to read Dr. Head's offensive remarks which appeared on page 476 of the October, 1956, issue of THE JOURNAL OF THORACIC SURGERY without saying to one's self "He's only joking," and letting it go at that. But since this form of wit (which used to be so prevalent in the past and which fortunately seems to be disappearing from the average surgeon's armamentarium) ceases to be funny in this era wherein those of us who are keenly interested in advancing the caliber of anesthesia practice are laboring to attract brighter minds to the specialty, it deserves some comment.

Dr. Head states that for the surgeon to relinquish the anesthetic care to another person would be violating his Hippocratic oath. May I point out that for something close to 80 years, following the discovery of ether, surgeons in general did a fairly thorough job of relinquishing this responsibility by paying almost no attention to the problems of disturbance of physiologic processes which occur under anesthesia. These studies awaited the appearance on the scene of an inquisitive group of physicians who called themselves anesthestists. Surgeons in fact went even one step further and allowed the important task of rendering patients unconscious to fall into the hands of ill-trained nurses and inexperienced doctors. I am told that one famous surgeon, perhaps even as famous as Dr. Head, used his chauffeur for his anesthestist. I would say that it would take something like a perverted sense of values to look upon such practices as carrying out one's Hippocratic responsibilities. On the contrary there seems to be a strong feeling going about that the successful carrying out of many surgical manipulations calls for the most intimate collaboration of surgeon and anesthetist, each performing his task independently and in concert, that it is a physical impossibility for the surgeon to perform both tasks well unless he has a very marked divergent squint and could move from the operating position to the head of the table with the speed of an electronic switching device. With all due respect to Dr. Head's fame as a surgeon, I doubt he is capable of doing that. In no branch of surgery has this intimate collaboration between the well-trained physicians in surgery and anesthsia proved itself more essential than in the field of thoracic surgery, where both are literally working in the same area. Dr. Head's suggestion that cardiac arrest is best prevented by a constant reference to blood being dark is hardly the answer to any question much less the serious question of the prevention of this catastrophe. It strikes me that it has been just the sort of practice which Dr. Head recommends which has been as responsible as anything else in failing to bring to light some of the important aspects of the alteration in human physiology under the stresses of anesthesia and surgery. We need clear and scientific minds for this problem, not minds warped by seeing this or that as an evil spirit.

Dr. Head's final remark was so important to my way of thinking that I wish he had said just this and nothing else. He refers to the need for special equipment for determining the oxygen saturation of the arterial blood, for monitoring $CO_2$ in inhaled mixtures and for a continuous perusal of the electrocardiogram, that all this should be the routine in good anesthesia practice. It is an interesting fact that many of these devices are on the market and could be used a good deal more routinely, at least in major intrathoracic operations. May I point out that most of these instruments are extremely expensive and can hardly be paid for in so much operating room feet. There is a crying need for reappraisal of the funds allocated for all the fancy trimmings which seem to be a part of the modern hospital with perhaps a greater allocation of funds for the purchase of these instruments which would tell us more about our patients. Now there is an evil spirit for Dr. Head to attack.

Very truly yours,
SEYMOUR SCHOTZ, M.D.
Chief, Department of Anesthesiology
The Presbyterian Hospital in Philadelphia

The Editor sent a copy of Dr Schotz's letter to Dr Head informing him that if he wished he could publish a reply Dr Head's reply is as follows

To the Editor of THE JOURNAL OF THORACIC SURGERY

I quite agree with you that the discussion of our papers should be open to discussion, and I would be very glad to have you print Dr Schotz's letter concerning my discussion I am very sorry that he took offense where no offense was intended I have reread my discussion and fail to see that it could reasonably give offense

I agree with Dr Schotz that the problem of cardiac arrest under anesthesia is a tremendously serious matter I disagree with him only in his statement that the specialty of anesthesiology is in need of brighter minds My experience with anesthesiologists has been very good and I am convinced that they have contributed importantly to the recent great advances in surgery However, they are only human, and when I see that the blood is dark, as I too frequently do even without turning my head toward the head of the table, I feel that I am justified in being alarmed and calling attention to it, and inasmuch as I am convinced that anoxia and hypercapnia are extremely dangerous, and hyperventilation is infinitely less dangerous, I feel justified in preferring to have my patients hyperventilated rather than hypoventilated Furthermore, I do not see how any surgeon can be other than deeply concerned about the fact that cardiac arrest during anesthesia has become one of the chief causes of death in surgery The patients come to the surgeon for treatment of their diseases, reasonably and legally he is the only one basically responsible to see that they come through the ordeal alive and well I believe that it is my attitude on this point which was chiefly offensive to Dr Schotz

If you decide to print Dr Schotz's letter, I would appreciate it very much if you would reprint my discussion and print this letter with it

Very sincerely yours,
JEROME R HEAD, M D
55 East Washington Street
Chicago 2, Illinois

# American Association for Thoracic Surgery

## CONSTITUTION

(As Amended to May 8, 1956)

### ARTICLE I. NAME

SECTION 1.—This Association shall be known as the American Association for Thoracic Surgery.

### ARTICLE II. OBJECT

SECTION 1.—The object of the Association shall be to encourage and stimulate investigation and study that will increase the knowledge of intrathoracic physiology, pathology, and therapy, to correlate such knowledge and disseminate it.

SECTION 2.—To attain this object, the Association shall hold at least one scientific meeting every year in which free discussion shall be featured; shall conduct a Journal for the publication of the papers presented at this meeting, and other acceptable articles; and shall undertake such other activities as the Council or the Association as a whole may decide.

### ARTICLE III. MEMBERSHIP

SECTION 1.—There shall be four classes of members: Active, Associate, Senior, and Honorary. Admission to membership in the Association shall be by election. There shall be no numerical limits on any class of membership. Only Active and Senior members shall have the privilege of voting or holding elective office.

SECTION 2.—Election to all classes of membership shall be for life, subject to the provisions of Section 3, following.

SECTION 3.—Membership may be voluntarily terminated at any time by members in good standing. The Council, acting as a Board of Censors, may recommend the expulsion of a member on the grounds of moral or professional delinquency, and submit his name, together with the grounds of complaint, to the Association as a whole at any of the regularly convened meetings, after giving the member so accused ample opportunity to appear in his own behalf.

### ARTICLE IV. OFFICERS AND GOVERNMENT

SECTION 1.—The officers of the Association shall be a President, a Vice-President, a Secretary, a Treasurer, an Editor, and five Councilors. These ten officers and councilors shall be the governing body of the Association, and shall have full power to act on all matters, except as follows:

1. They may not alter the initiation fees or annual dues, nor levy any general assessments against the membership, except that they may, in individual cases, remit annual dues or assessments.

2. They may in no wise change the Constitution or By-Laws.

3. They may neither elect new members nor alter the status of existing members, other than to apply the provisions of Article III, Section 3.

4. They may not deplete the principal of the Endowment Fund.

SECTION 2.—Officers and Councilors shall be elected at the annual meeting of the Association, and shall take office upon conclusion of the meeting. The President and the Vice-President shall be elected for a one-year term of office and neither may be re-elected to succeed himself in the same office. The Secretary, the Treasurer, and the Editor shall be elected for a one-year term of office, and any or all may be re-elected indefinitely. The outgoing President shall automatically become a Councilor for a one-year term of office. The other four Councilors shall be elected, one each year, for a four-year term of office, but no Councilor may be re-elected to succeed himself.

SECTION 3.—Vacancies occurring among the officers and councilors during the year shall be temporarily filled by action of the Council, subject to approval of the Association at the next regularly convened meeting.

## ARTICLE V. COMMITTEES

SECTION 1.—At the opening session of the annual meeting there shall be elected, after nomination from the floor of the Association, a Nominating Committee of three. This Committee shall prepare a slate of nominees for officers and councilors and shall present their report at the executive session of the Association.

SECTION 2.—The Council is empowered to appoint a Membership Committee, an Auditing Committee, a Program Committee, a Necrology Committee, and such other committees as may in its opinion be necessary. All such committees shall render their report at the executive session of the Association.

SECTION 3.—The Editor is empowered to appoint an Editorial Board, subject only to the approval of the Council.

SECTION 4.—The Association as a whole may authorize the Council to appoint Scientific or Research Committees for the purpose of investigating thoracic problems and may further authorize the Council to support financially such committees to a limited degree. In appointing such committees, the Council shall be governed by the provisions of the By-Laws.

## ARTICLE VI. FINANCES

SECTION 1.—The fiscal year of the Association shall run from the beginning of one annual meeting to the beginning of the next annual meeting. The books of the Association shall be kept and audited on this basis.

SECTION 2.—Members shall contribute to the financial maintenance of the Association through the medium of initiation fees, annual dues, and special assessments. The amount of the annual dues and the initiation fees shall be determined by the By-Laws.

If, at the end of any fiscal year, there be a deficit in the current funds of the Association, the Council may send out notices to that effect tive Members to contribute the necessary amount so that no deficit be carried over from one fiscal year to another. The Association may, in any regularly convened meeting, vote a special assessment for any purpose consistent with the objects of the Association (Article II), and such special assessment shall become an obligatory charge against the classes of members affected thereby.

SECTION 3.—To meet the current expenses of the Association, there shall be available all revenue derived from annual dues, special assessments, and income from Endowment Fund, subject to the provisions of Section 4, following. Funds derived from the payment of initiation fees shall not be available for current expenses.

SECTION 4.—All funds derived from the payment of initiation fees shall be placed in a special fund, to be invested and reinvested in legal securities, to be held intact, and to be known as the Endowment Fund. The Council is responsible for the proper management of the Endowment Fund, and may divert any surplus in the current funds of the Association into this fund, but may not withdraw any of the principal of the Endowment Fund except in accordance with the provisions of Section 6, following.

SECTION 5.—The income from the Endowment Fund shall be expended as the Council directs.

SECTION 6.—The principal of the Endowment Fund may be withdrawn, in whole or in part, under the following conditions only: The amount of principal to be withdrawn shall have been approved by the Council; it shall have been approved by a majority of the members present and voting at a regularly convened annual meeting; it shall have been tabled for one year; it shall have been finally passed by a three-fourths vote of the members present and voting at the next regularly convened annual meeting.

SECTION 7.—In the event of the dissolution of the Association, the Endowment Fund shall be distributed among national institutions of the United States and Canada in a proportion equal to the then existing ratio between the numbers of citizens of the two nations who are members of the Association.

## ARTICLE VII. MEETINGS

SECTION 1.—The time, place, duration, and procedure of the annual meeting of the Association shall be determined by the Council, and the provisions of the By-Laws.

SECTION 2.—A special meeting of the Association may be called on one month's notice on the written request of fifteen members. The specific purposes of the meeting must be stated in the request and in the official call for the meeting.

SECTION 3.—The annual meeting of the Council shall be held at or near the close of the fiscal year.

## ARTICLE VIII. AMENDMENTS

SECTION 1.—This Constitution shall in no wise be changed except by a three-fourths vote of the members present at an annual meeting, and further provided that the proposed alteration or amendment shall have been moved and seconded at a previous annual meeting and that printed copies of the suggested alteration or amendment shall have been circulated among the members, and that the members shall have been specifically advised that such alteration or amendment will be voted upon

## BY-LAWS

### ARTICLE I

SECTION 1—These By-Laws shall merely interpret the Constitution and specifically apply its principles They shall set forth no principles not included in the Constitution

### ARTICLE II

SECTION 1—All papers read before the Association shall become the property of the Association Authors shall leave original copies of their manuscripts with the Editor or Reporter, at the time of presentation, for publication in the official journal

SECTION 2—When the number of papers makes it desirable, the Council may require authors to present their papers in abstract, and may set a time limit on discussions

SECTION 3—Members are urged to cooperate with all Scientific Committees of the Association

SECTION 4—Attendance at Annual Meetings and participation in the scientific programs shall be optional for all Honorary and Senior Members but it shall be expected from all Active and Associate Members

SECTION 5—While the scientific session of the annual meeting is held primarily for the benefit of the members of the Association, it may be thrown open to nonmembers who are able to submit satisfactory credentials, who register in a specified manner, and who pay such registration fee as may be determined and published by the Council from year to year

### ARTICLE III

SECTION 1—Candidates for membership in this Association must be formally nominated and seconded, in an approved manner, by not less than three Active or Senior Members Such nomination must have been in the hands of the Membership Committee for not less than four months, and the name of the candidate must have been distributed to the Association as a whole before final action may be taken on any new candidate for election to Active or Associate Membership Provided the foregoing requirements have been met, and the candidates have been approved by the Membership Committee and by the Council, their names shall be presented to the Association at a regularly convened annual meeting for final action A three fourths vote of those present and voting shall be required to elect Any candidate for membership in this Association who has failed of election for two successive years shall automatically cease to be a candidate and may not be renominated until after a lapse of three years.

SECTION 2.—There shall be no numerical limit on Active Membership. The candidate, to be eligible, must be a citizen of the United States of America or of Canada, unless in unusual cases this citizenship requirement shall have been waived by the Council. The candidate shall have achieved distinction in the thoracic field, or shall have made a meritorious contribution to knowledge pertaining to thoracic disease or its surgical treatment. Under usual circumstances, Active Members shall be elected from the Associate Members who have satisfied all requirements for Active Membership. Under unusual circumstances, Active Membership may be awarded to new candidates of outstanding distinction.

SECTION 3.—There shall be no numerical limit on Associate Membership. The candidate, to be eligible, may be a citizen of any country. All candidates for Associate Membership shall be engaged actively in the practice of clinical medicine, or in teaching, or investigative work in the basic sciences underlying clinical practice, and all shall have demonstrated an active interest in thoracic disease. In the case of those who are engaged in the independent practice of thoracic surgery, it shall be required that they present evidence of sound basic training in general as well as thoracic surgery. Clinicians of outstanding ability, whose interest is limited to a small fraction of the entire thoracic field, may be elected to Associate Membership.

SECTION 4.—The number of Senior Members shall be unlimited. The candidate, to be eligible, must have been an Active Member of the Association for not less than ten years or, having been an active Member, have passed the age of sixty years, or become incapacitated by illness.

SECTION 5.—Honorary Membership shall be reserved for such distinguished persons as may be deemed worthy of this honor by the Council with concurrence of the Association.

SECTION 6.—The report of the Membership Committee shall be rendered at the annual Executive Session of the Association. Candidates shall be presented in groups in the following order: Candidates for Honorary Membership; retirement of Active Members to Senior Membership; candidates for Active Membership; candidates for Associate Membership.

SECTION 7.—The Council shall recommend that any Active or Associate member whose dues are in arrears for two years, or who has been absent, without sufficient excuse, from three consecutive annual meetings, shall have his membership terminated.

SECTION 8.—Notwithstanding Section 7, any member of the Association over 60 years of age is excused from the attendance requirement and upon his specific request may likewise be excused from the payment of dues.

## ARTICLE IV.

SECTION 1.—The President of the Association shall perform all duties customarily pertaining to the office of President. He shall not only preside at all meetings of the Association, but also at all meetings of the Council. The President shall be elected from the Active Members of the Association.

SECTION 2.—The Vice-President of the Association shall perform all duties customarily pertaining to the office of the Vice-President, not only as to the Association, but also as to the Council. The Vice-President shall be elected from the Active Members of the Association.

SECTION 3.—The Secretary of the Association shall perform all duties customarily pertaining to the office of Secretary. He shall serve not only as Secretary of the Association but also as Secretary of the Council. The Secretary shall be elected from the Active Members of the Association.

SECTION 4.—The Treasurer of the Association shall perform all duties pertaining to the office of Treasurer. He shall not only serve as Treasurer of the Association but shall also serve as custodian of the Endowment Fund. The Treasurer shall be elected from the Active Members of the Association.

SECTION 5.—The Editor of the Association shall be the Editor of the official Journal and shall, ex officio, be the Chairman of the Editorial Board. The Editor shall be elected from the Honorary, Active, or Senior Members of the Association.

SECTION 6.—The Councilors of the Association shall hold office as specified in the Constitution. They may be elected from either the Active Members or the Senior Members of the Association.

SECTION 7.—In the event of a vacancy occurring in the office of President, the Council shall advance the Vice-President to the Presidency and appoint a new Vice-President under the Provisions of Article IV, Section 3, of the Constitution.

### ARTICLE V.

SECTION 1.—The Nominating Committee shall consist of three Active or Senior Members elected in accordance with the provisions of Article V, Section 1, of the Constitution. The Council shall instruct the Committee as to the vacancies which are to be filled by election.

SECTION 2.—The Membership Committee shall consist of seven Active or Senior Members appointed in accordance with the provisions of Article V, Section 2, of the Constitution. The Council may appoint not more than one of its own members to serve on this Committee. The duties of the Membership Committee are to investigate all candidates for membership in the Association and to report their findings as expeditiously as possible to the Council through the Secretary of the Association. This Committee is also charged with searching the literature of this and other countries to the end that proper candidates may be presented to the Association for consideration. Appointment to this Committee shall be for a period of one year, and not more than five of the members may be reappointed to succeed themselves. This Committee is also charged with maintaining a record of membership attendance and participation in the scientific programs and reporting to the affected members and to the Council any deviations from the requirement of Article II, Section 4, of these By-Laws.

SECTION 3.—The Auditing Committee shall consist of three Active or Senior Members appointed in accordance with the provisions of Article V, Section 2, of the Constitution. None of these may be selected from the officers or councilors of the Association. Their duty shall be to audit the accounts of the Association at the end of each fiscal year and render their report to the Council of the Association through the Secretary. Appointments to this Committee shall be made for a one-year term. Not more than two members may be reappointed to succeed themselves.

SECTION 4.—The Program Committee shall consist of five members: The President of the Association, the Secretary of the Association, the Editor of the Association, and two members at large. one of whom shall be resident at or near the place of annual meeting. The duties of this Committee shall be to arrange, in conformity with instructions from the Council, the scientific program for the annual meeting.

SECTION 5.—The Necrology Committee shall consist of one or more Active or Senior Members, and shall be appointed in accordance with the provisions of Article V, Section 2. of the Constitution. Appointments to this Committee shall be for a one-year term of office. Any or all members of this Committee may be reappointed to succeed themselves. The Council may, if it so desires, appoint one of its own members to serve as Chairman of this Committee. The duties of the Necrology Committee shall be to prepare suitable resolutions and memorials upon the deaths of all members of the Association and to report such deaths at every annual meeting.

SECTION 6.—The Editorial Board shall be appointed by the Editor, subject only to the approval of the Council. The Editor shall be. ex officio, the chairman of this board and shall be privileged to appoint and indefinitely reappoint such members of the Association. regardless of class of membership, and such nonmembers of the Association as in his opinion may be best calculated to meet the editorial requirements of the Association.

SECTION 7.—When Scientific or Research Committees are authorized by the Association, the Council shall appoint the Chairmen of these Committees, with power to organize their committees in any way best calculated to accomplish the desired object, subject only to the approval of the Council. Financial aid rendered to such Committees shall not exceed such annual or special appropriations as may be specifically voted for such purposes by the Association as a whole.

### ARTICLE VI.

SECTION 1.—Honorary Members of the Association are exempt from all initiation fees, dues. and assessments.

SECTION 2.—Annual dues for Active Members shall be $30.00.

SECTION 3.—Annual dues for Associate Members shall be $25.00.

SECTION 4.—Senior Members are exempt from dues.

SECTION 5.—Initiation fee for those elected directly to Active Membership shall be $15.00.

SECTION 6.—Initiation fee for those elected to Associate Membership shall be $10.00. If and when an Associate Member is elected to Active Membership, he shall pay an additional $5.00 initiation fee.

SECTION 7.—Income from the Endowment Fund shall be expended as the Council directs.

SECTION 8.—Associate and Active Members must subscribe to THE JOURNAL OF THORACIC SURGERY to retain their membership status.

SECTION 9.—Senior Members may retain their membership status without the payment of annual dues and subscription to THE JOURNAL OF THORACIC SURGERY is optional.

(Note.—Bills for membership dues and for subscriptions to THE JOURNAL OF THORACIC SURGERY *will be mailed to members by the Treasurer after the Annual Meeting.*)

## ARTICLE VII.

SECTION 1.—When the Association convenes for its annual meeting, it shall immediately go into executive session, but the business at this session shall be limited to:

1. Election of Nominating Committee.
2. Appointment of necessary committees.
3. Miscellaneous business of an urgent nature.

SECTION 2.—The annual executive session of the Association shall be held at the opening of the afternoon session of the second day of the meeting. The order of business shall be:

1. Reading of the minutes of the preceding meetings of the Association and Council.
2. Report of the Treasurer for the last fiscal year.
3. Report of the Auditing Committee.
4. Report of the Treasurer for the current year to date.
5. Report of the Necrology Committee.
6. Report of the Program Committee.
7. Action on amendments to the Constitution and By-Laws.
8. Action on recommendations emanating from the Council.
9. Unfinished Business.
10. New Business.
11. Report of the Membership Committee.
12. Election of new members.
13. Report of the Nominating Committee.
14. Election of officers.

SECTION 3.—At the annual meeting of the Council the order of business shall be:

1. Reading of minutes.
2. Treasurer's report.

3. Appointment of committees.
4. Determination of instructions to be given to Auditing, Membership, Necrology, and other committees except Program Committee.
5. Determination of details in connection with the annual meeting.
6. Appointment and instructions to the Program Committee.
7. Unfinished Business.
8. New Business.

SECTION 4.—There shall be an annual meeting of the Council.

## ARTICLE VIII.

SECTION 1.—These By-Laws shall in no wise be changed except by a two-thirds vote of the members present at the annual meeting or a properly convened meeting of the Association, and further provided that the proposed action or amendment shall have been moved and seconded by not less than three of the members in a properly convened annual or special meeting of the Association.

SECTION 2.—These By-Laws may be suspended in whole or in part for a period of not more than twelve hours by a unanimous vote of those present at any regularly convened meeting of the Association.

# The Journal of Thoracic Surgery

---

Vol 33 | February, 1957 | No 2

---

## Original Communications

## ROENTGEN CONTRAST DIAGNOSIS OF EXPERIMENTAL MITRAL AND AORTIC INSUFFICIENCY IN DOGS BY TRANSVENTRICULAR INJECTION AND RETROGRADE CATHETERIZATION

Robert J Wilder, M D ,* Howard L Moscovitz, M D , and
Mark M Ravitch, M D **
New York, N Y

Recent advances in cardiac surgery have served to emphasize the need for greater precision in the clinical diagnosis of valvular lesions of the heart Physical diagnostic methods, electrocardiography, electrokymography, cardiac catheterization, and even direct pressure tracings from the heart[1] may all fail to disclose the existence of significant mitral insufficiency, particularly when this lesion is combined with mitral stenosis The surgeon's finger itself may in some instances be incapable of estimating the degree of mitral insufficiency [2] With the steady increase in the amount of aortic valvular surgery being performed, the more accurate demonstration of the anatomic and functional details of aortic valve lesions becomes essential

Experimental visualization of the dog ventricle by direct injection of contrast medium was performed first in 1933 by Reboul and Racine,[3] utilizing an intercostal needle In 1936, Nuvoli,[4] using the same approach, injected sodium iodide solution into the left ventricle of a patient with an aortic aneurysm In 1945, Radner[5] visualized the coronary arteries in man by direct puncture of the ascending aorta through the sternum In 1951, Nuñez and Ponsdomenech[6, 7] coined the term "cardioangiography" for the direct injection of contrast media into the chambers of the human heart They studied 30 patients in this manner without mortality and made the diagnosis of mitral insufficiency when radiopaque material was noted to reflux into the

---

From the Departments of Surgery and Medicine of Mt Sinai Hospital New York
Received for publication Dec 19 1955

*This work was done during Dr Wilder s tenure of a Fellowship of the New York Heart Association Present address Baltimore City Hospitals Baltimore Md

**Present address Baltimore City Hospitals Baltimore Md

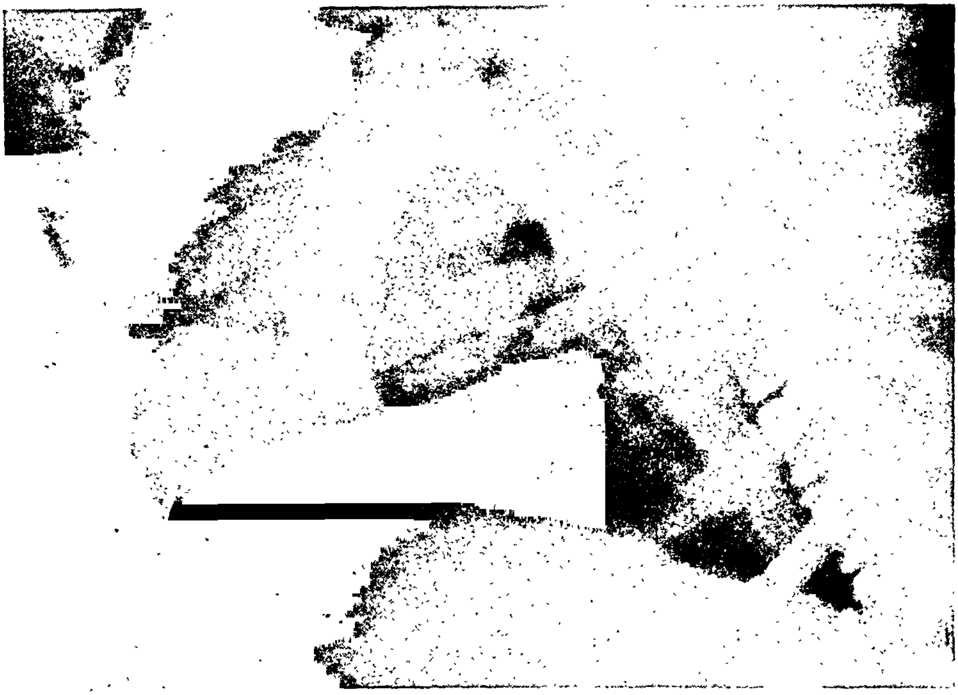


Fig. 1.—Roentgenogram following direct needle puncture and injection of Urokon into left ventricle of a normal dog. The needle is seen entering the left ventricle from below. The competent mitral valve prevents regurgitation of the contrast medium into the left atrium.

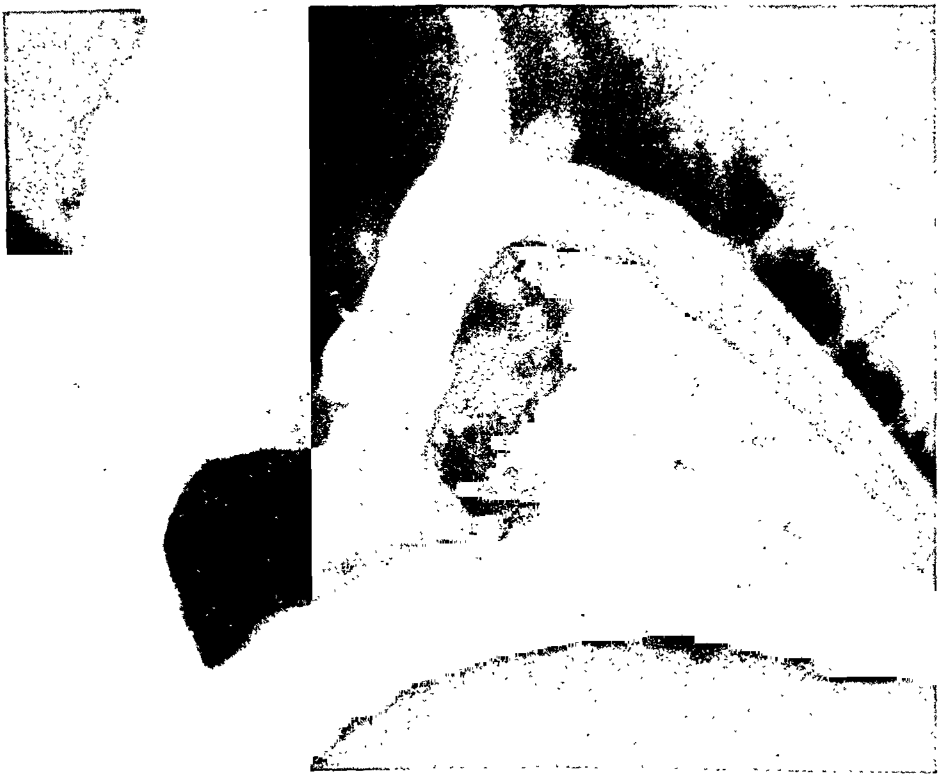


Fig. 2.—Retrograde catheterization of the left ventricle of a normal dog and dye injection through a No. 9 Lehman aortic catheter. The left ventricle, mitral valve area, sinuses of Valsalva, and a coronary artery branch are visualized. No dye has refluxed into the left atrium.

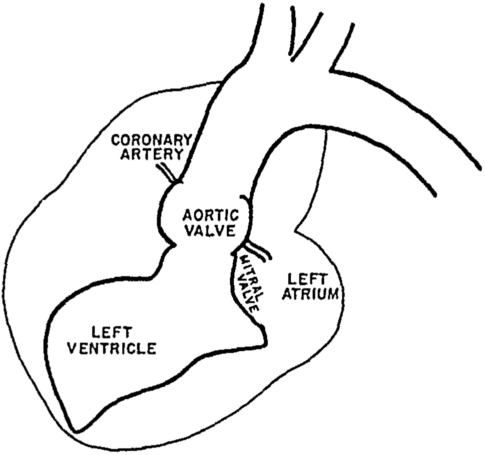


Fig 3 —Diagrammatic representation of anatomic details of roentgenogram shown in Fig 2.

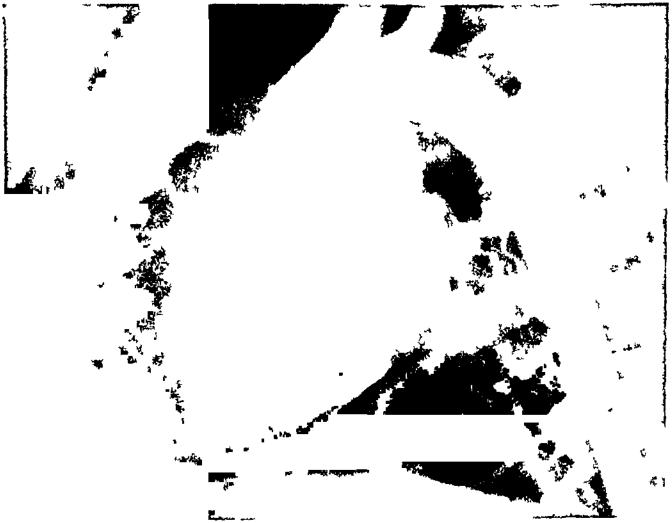


Fig 4 —The anatomy of the normal dog aortic valve and arch following injection of 70 per cent Urokon through a retrograde catheter with tip in ascending aorta No dye is seen in the left ventricle

border. This margin represents the closed mitral valve. The normal competent mitral valve prevents visualization of the left atrium which lies just posterior to this sharp crescent-shaped border. In this projection, the valve lies so that the posteromedial commissure is at the base of the aorta and the anterolateral commissure opposite. The outline of the ventricle is sharp and the entire ventricle is filled with contrast medium. The contour of the ventricular chamber differs depending on whether the film is taken in systole or diastole. Visualization of the aortic valve leaflets and the bulging sinuses of Valsalva is especially clear.

When radiopaque material is injected into the ascending aorta through an arterial catheter, the sinuses of Valsalva, the coronary arteries, and the aortic valve leaflets are sharply visualized. No dye is seen in the left ventricle (Fig. 4). In a series of 19 injections of contrast medium into the left ventricle by both techniques, and ten injections of contrast medium into the ascending aorta by retrograde arterial catheterization, complete competency of the mitral and aortic valves in normal dogs was demonstrated.

*The Heart in Mitral Insufficiency.*—In 20 of the 38 dogs studied, mitral insufficiency was created as described and radiographic studies recorded. Fig. 5, *A* and *B*, demonstrates the results of direct and of retrograde injection of contrast medium into the left ventricle of 2 dogs with experimental mitral insufficiency. Because of the damaged mitral valve, the dye regurgitates freely into the left auricle. The retrograde arterial catheter makes it possible to inject a greater volume of dye more rapidly than by the needle technique, and a denser roentgenogram results.

*Chronic Mitral Insufficiency.*—Serial x-rays obtained over a period of one year in a dog with chronic mitral insufficiency are illustrated in Fig. 6. The increasing size of the auricle is apparent by either of the two techniques of injection. In some animals, the insufficient mitral valve in time becomes competent once again by the heaping up of granulation and fibrous tissue at the edges of the operative lesion. Fig. 7 demonstrates the x-ray films of a dog, 2 months, 5 months, and 7 months after the creation of a small mitral insufficiency. The x-ray at 7 months was interpreted as showing no regurgitation and the dog was then re-explored. No apparent regurgitation could be detected at that time either by digital examination or by pressure recordings from the left atrium. Post-mortem examination revealed the lesion in the posterior leaf of the mitral valve to be healed by fibrosis around the edges of the previous valve rupture. Fig. 8 illustrates examples of marked mitral insufficiency of 6 months' duration in 2 dogs using both techniques of injection.

The group of 20 dogs, with experimentally produced mitral insufficiency, were followed with serial radiographic studies at two- to three-month intervals by direct and retrograde injection of contrast material. In all cases, where two or more successive x-rays revealed that mitral insufficiency no longer existed, a second operation confirmed the presence of a competent valve. There were 3 dogs in which two consecutive x-rays were inconclusive. At operation a small regurgitant jet could be intermittently felt in 1 and a small but definite

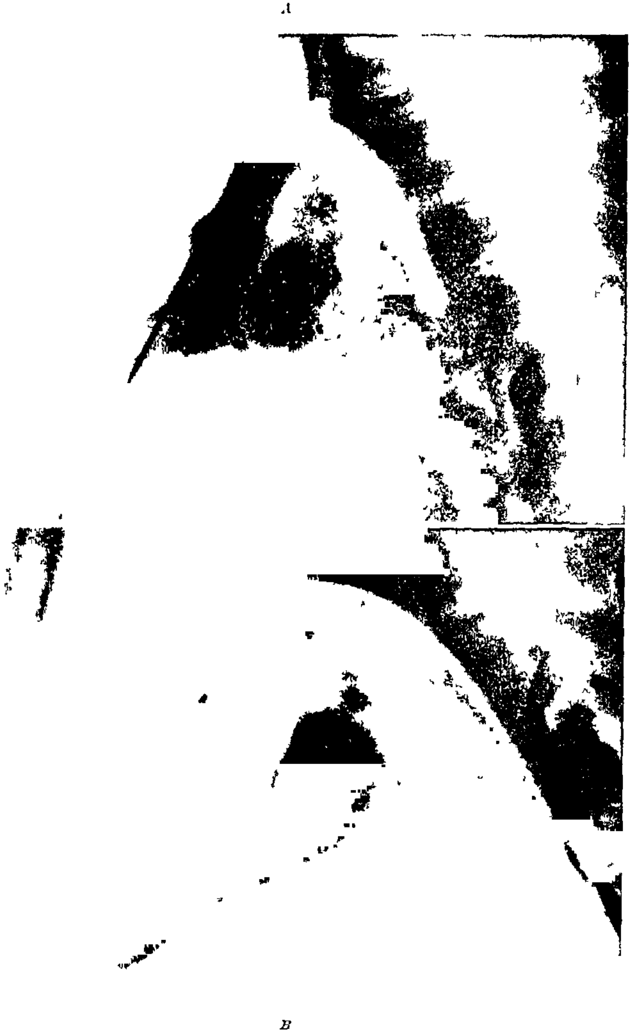


Fig 5 —The roentgen appearance of the dog heart with experimentally produced mitral insufficiency Free reflux of dye into the left atrium through the incompetent mitral valve is seen after left ventricular injection *A*, direct needle puncture of left ventricle *B*, retrograde catheterization of left ventricle

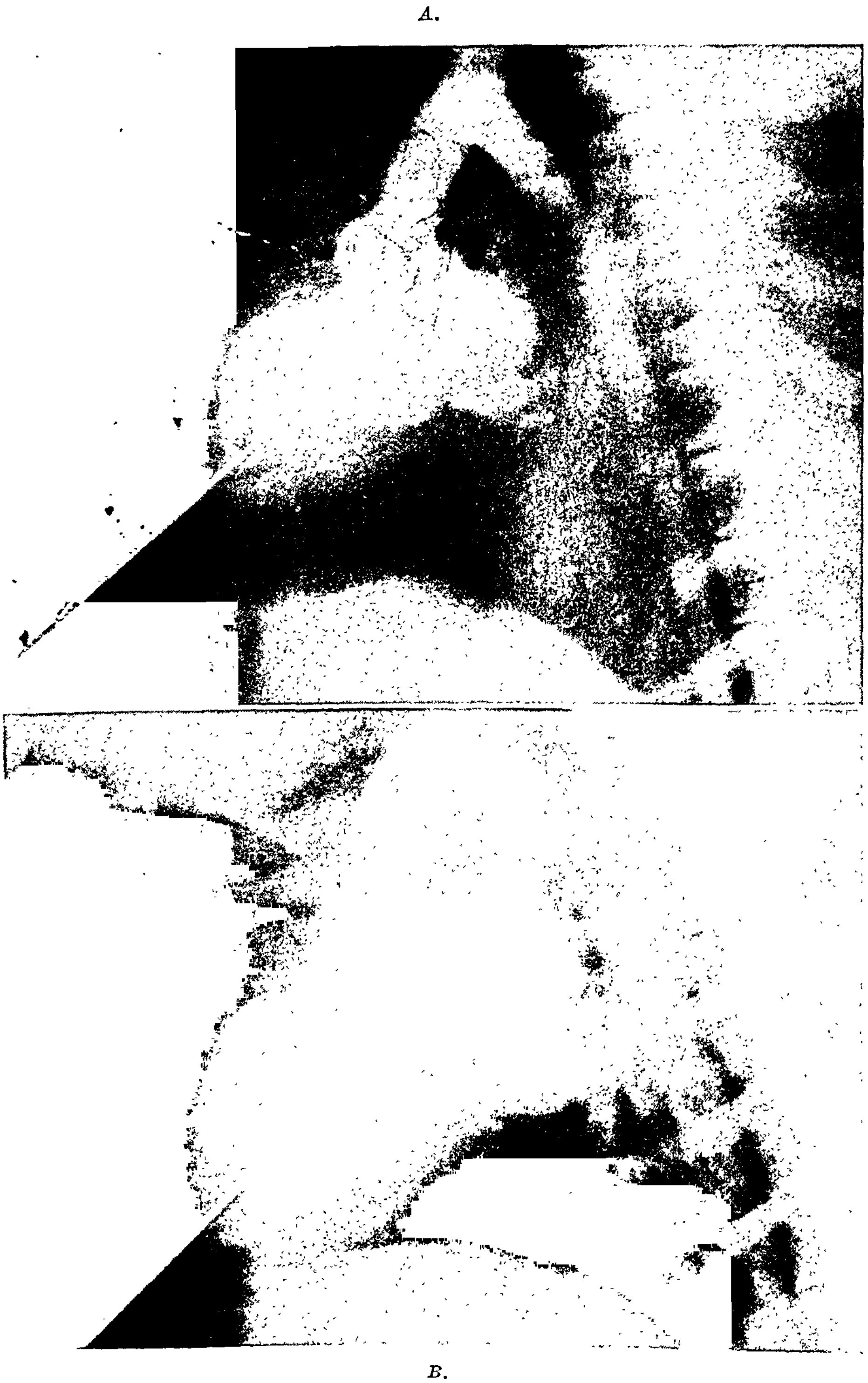
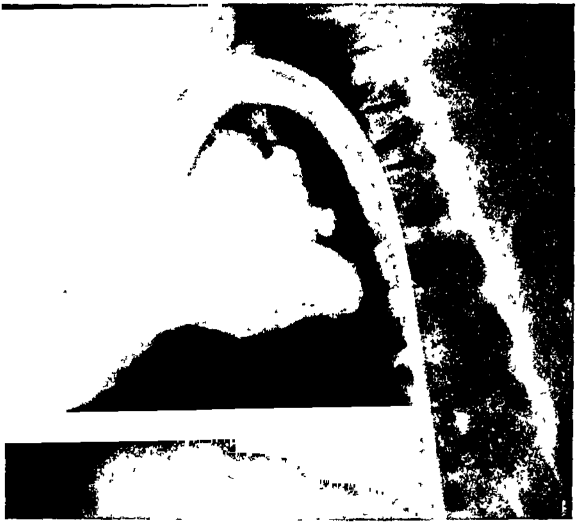


Fig. 6.—A series of left ventricular dye injections in a dog with chronic mitral insufficiency over the course of one year. Note the progressive left atrial and pulmonary vein dilatation. *A*, postoperatively; *B*, 6 months later.

regurgitant jet could be felt in 2. There was no obvious change in the left atrial pressure curve in any of these 3 cases. Of the other 17 dogs, 4 had no mitral insufficiency, two to six months postoperatively, as demonstrated by x-ray and confirmed at re-exploration. There were 13 dogs with varying degrees of insufficiency demonstrable by dye injection, which persisted for as long as 15 months after the creation of mitral insufficiency.

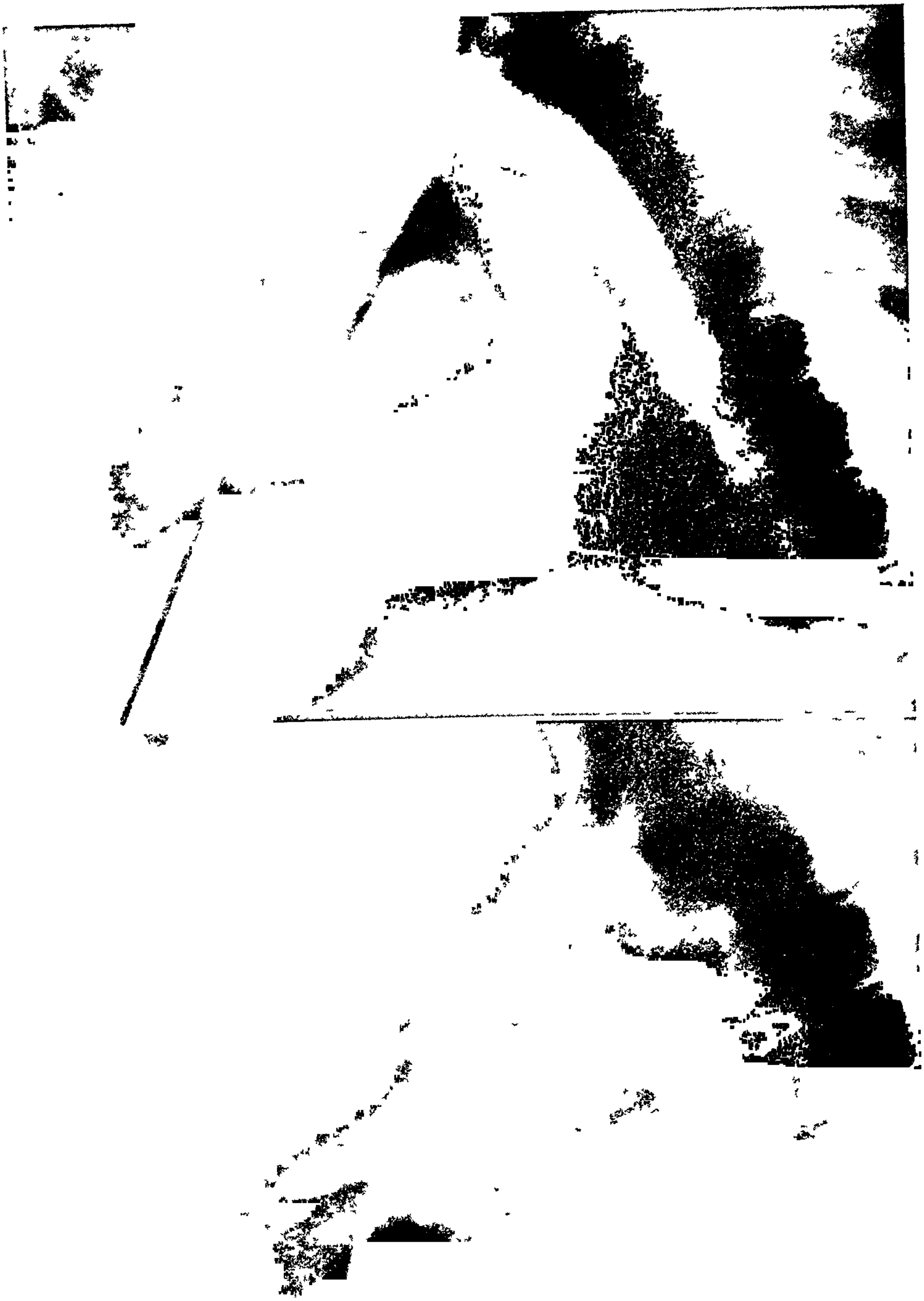


*C.*

Fig. 6—Cont'd. *C*, 1 year later.

*Mitral Insufficiency and Mitral Stenosis.*—The double valvular lesion of mitral stenosis and mitral insufficiency was produced and x-rays recorded in the manner described. Fig. 9 illustrates the roentgen appearance of the left cardiac chambers in dogs with combined mitral stenosis and mitral insufficiency. The size of the mitral orifice and the position of the experimental stenosis may be estimated from the films in many cases. In Fig. 9, *A*, the stenosing suture was passed around the atrium above the mitral annulus and the left circumflex coronary artery. In this film the constriction of the atrium just above the annulus is obvious. The stenosis is of moderate degree. Fig. 9, *B*, reveals the x-ray in a dog with severe mitral stenosis and moderate insufficiency. In this case, the stenosing suture was tightened in a plane just below the left circumflex coronary artery. The narrowing of the mitral valve area is clearly visualized. The left circumflex coronary artery appears to be uninvolved by the circumflex suture. Pressure tracings recorded at the time of the surgical production of mitral stenosis revealed evidence of a markedly elevated left atrioventricular diastolic pressure gradient. In Fig. 10, *A*, the x-ray reveals a

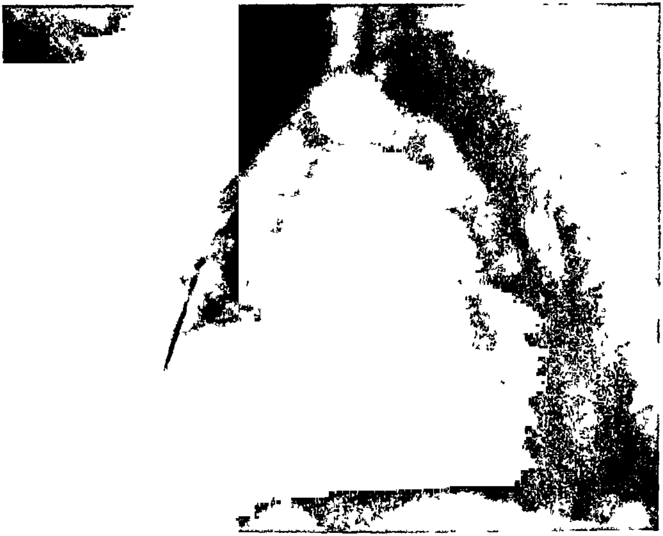
*A.*



*B.*

Fig. 7.—Spontaneous healing of experimentally produced mitral insufficiency. *A*, Two months postoperatively; *B*, 5 months postoperatively.

significant mitral insufficiency. In this animal, at a second operation, a stenosing suture was passed and tightened around the mitral ring below the annulus. A subsequent dye injection revealed apparent reduction in the amount of mitral regurgitation (Fig. 10, *B*). It should be noted that when a circumferential stenosing suture is passed around the mitral ring below the annulus, the course of the coronary artery may occasionally be modified and in Fig. 10, *B*, the buckling of the circumflex coronary artery is obvious.



*C.*

Fig. 7.—Cont'd. *C*, 7 months postoperatively. The regurgitant jet of contrast medium has disappeared 7 months after the lesion was created. No mitral insufficiency could be detected at re-exploration or at post-mortem

*Aortic Insufficiency.*—The normal aortic valve was studied by retrograde aortic catheterization and dye injection in 10 dogs. In 5 dogs, aortic insufficiency was produced and studied by the same technique. With a competent aortic valve as shown in Fig. 3, dye introduced into the ascending aorta outlines the aortic valve, sinuses of Valsalva, and the coronary arteries. No dye is seen in the left ventricle. Fig. 11 demonstrates the appearance of the roentgenogram in a dog with aortic insufficiency. Free reflux of the contrast medium into the left ventricle has taken place.

*Cine-angiography.*—The image intensifier makes it possible to record high-speed motion pictures of a fluoroscopic screen at the rate of 48 frames per second during the injection of radiopaque media into the ascending aorta, the left ventricle, and the left atrium. These motion pictures demonstrate the dynamic details of valvular anatomy and function both in the normal and in the experimentally altered state. The flow of dye from one cardiac chamber to another

Fig. [illegible] [illegible]

is clearly visualized as well as the motion of the valve leaflets. The mixing and diluting of the dye with blood graphically demonstrates the action of eddy currents in the heart chambers. Lesser degrees of aortic and mitral insufficiencies may be detected in this manner and this technique may yield additional information in localizing and determining the size of various valvular lesions.

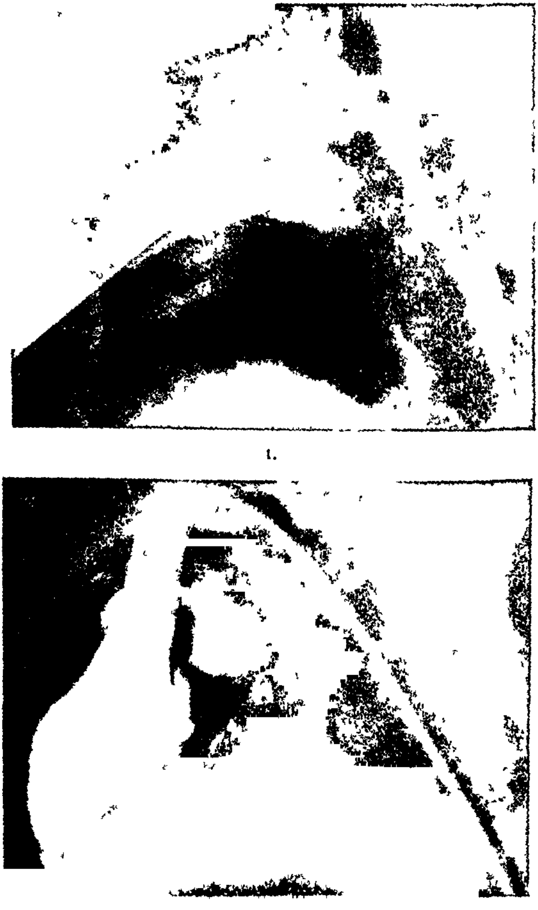


Fig 9.—Mitral stenosis and insufficiency. *A*, The stenosing circumferential mitral suture has moderately narrowed the left atrium above the mitral annulus. *B*, The suture has been passed just below the mitral annulus and has produced a high grade of stenosis. Mitral regurgitation is still readily visible despite the marked narrowing of the mitral orifice.

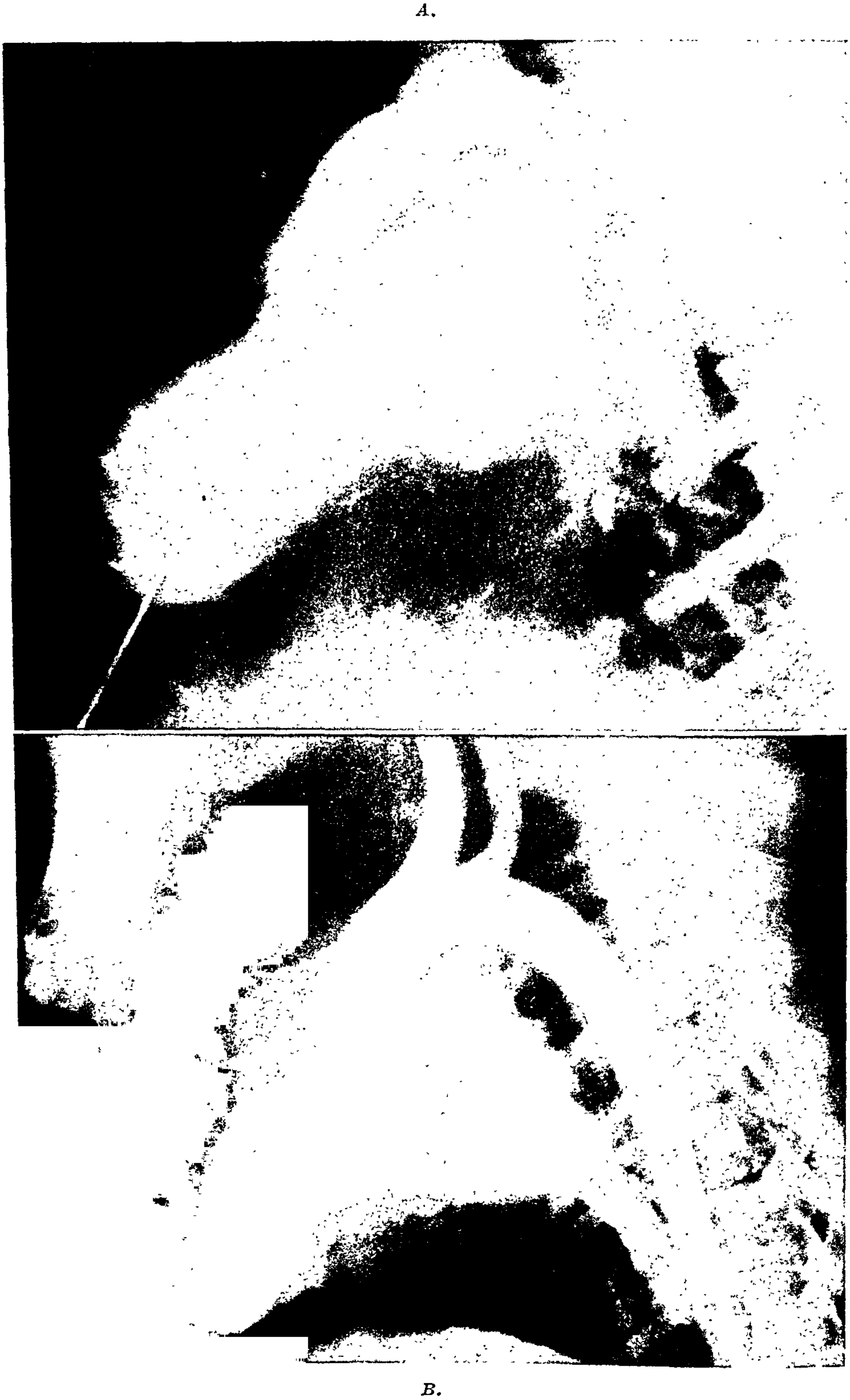
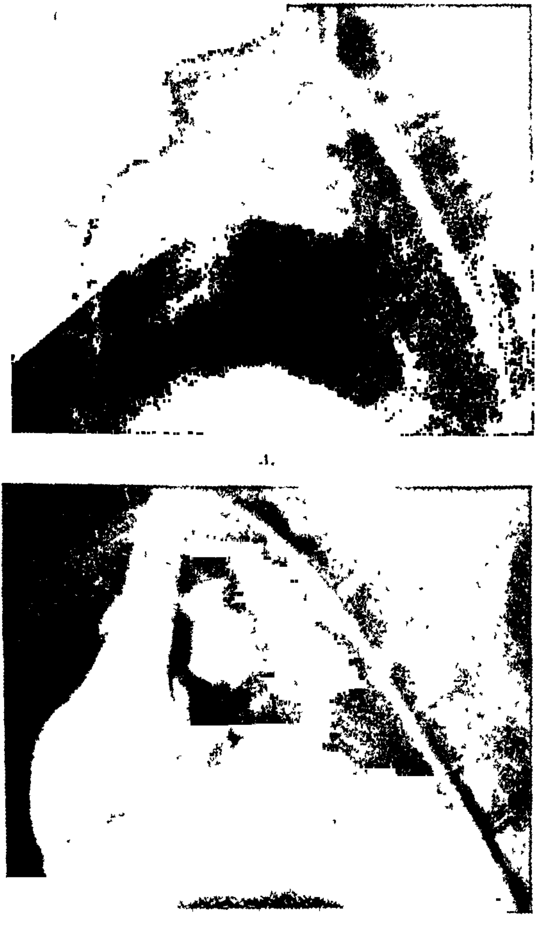


Fig. 8.—Chronic mitral insufficiency of 6 months' duration. The considerable enlargement of the left atrium and dilatation of the pulmonary veins is apparent.

is clearly visualized as well as the motion of the valve leaflets The mixing and diluting of the dye with blood graphically demonstrates the action of eddy currents in the heart chambers Lesser degrees of aortic and mitral insufficiencies may be detected in this manner and this technique may yield additional information in localizing and determining the size of various valvular lesions.



A.

B.

Fig. 9 —Mitral stenosis and insufficiency *A*, The stenosing circumferential mitral suture has moderately narrowed the left atrium above the mitral annulus *B*, The suture has been passed just below the mitral annulus and has produced a high grade of stenosis. Mitral regurgitation is still readily visible despite the marked narrowing of the mitral orifice

*Complications.*—In the over-all series of 38 dogs, no dog developed any permanent deleterious effect following a single intraventricular injection of the radiopaque medium. Bursts of premature ventricular contractions characteristically occurred at the time of the injection with a transient drop in blood pressure. However, when repeated and closely spaced injections were carried out, the dogs frequently developed serious arrhythmias, hypotension, and, in some cases, died.

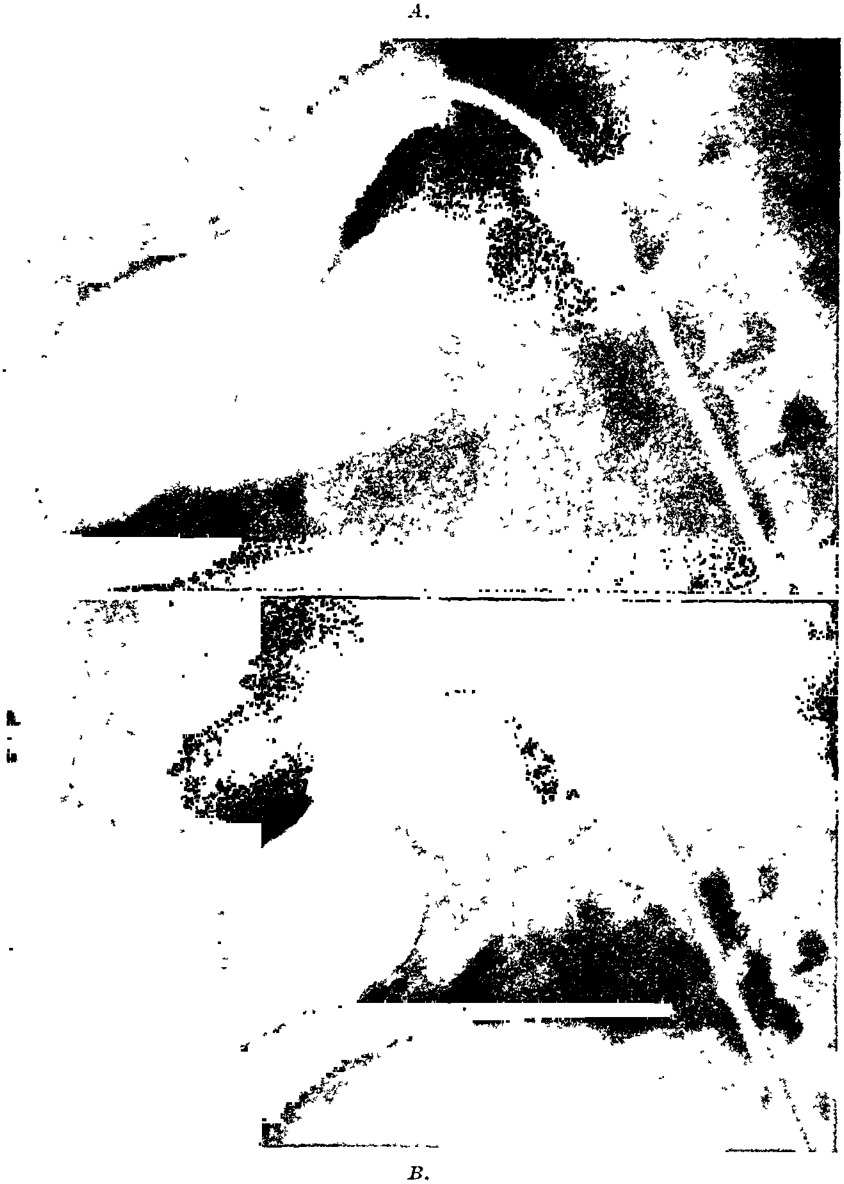


Fig. 10.—Mitral insufficiency and stenosis. *A*, The lesion of mitral insufficiency alone shown. *B*, Two months later a moderately tight stenosing suture has been superimposed the incompetent mitral valve. An apparent reduction in the degree of mitral regurgitation has taken place. Note the tortuosity of the left circumflex coronary artery produced by the mitral stenosis.

Approximately 91 direct injections and 20 retrograde injections into the left ventricle were carried out in the colony of 20 dogs with mitral insufficiency. These dogs were usually subjected to only one or two injections of contrast medium during any one 24-hour period. In this long-term series, there were two fatalities with the direct injection technique and none using the retrograde catheter. The first fatality took place when the needle slipped back into the wall of the heart and 5 to 10 c.c. of dye was injected into the left ventricular myocardium (Fig. 12, *A*). The dog suffered a convulsion and died immediately. The second dog was found dead 30 minutes after the cardiac puncture and injection. Post-mortem examination revealed several hundred cubic centimeters of blood in the pleural cavities. No laceration could be found in the ventricular wall or in a coronary vessel to explain the bilateral hemothorax. The pericardium had been left widely open several months earlier at the creation of mitral insufficiency. In 2 other dogs, small amounts of dye in the ventricular myocardium were seen on the films although nothing untoward had been noted during the injection and both dogs survived.

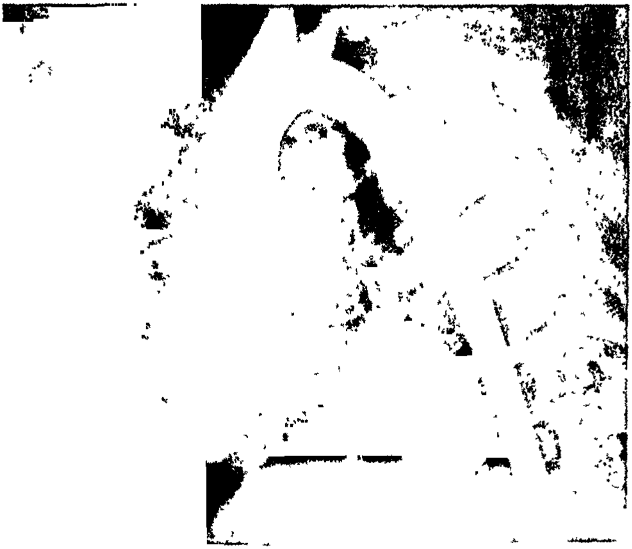
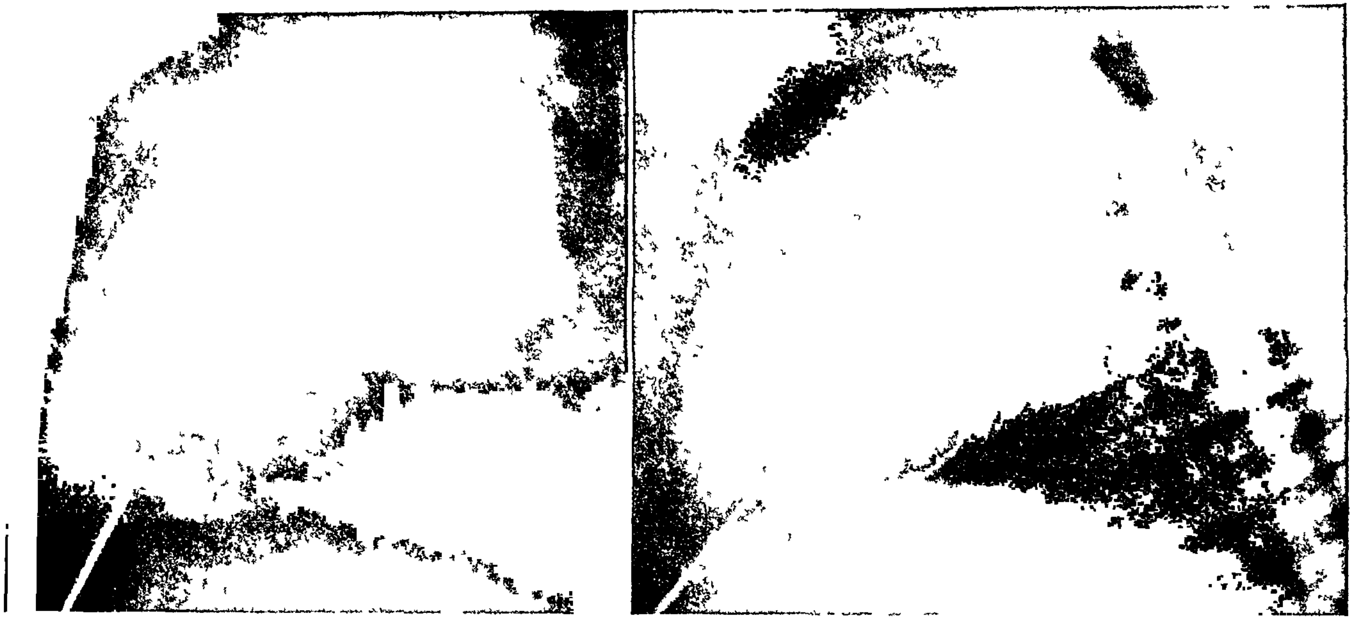


Fig. 11.—Aortic insufficiency. Dye injected into the ascending aorta freely regurgitates into the *left* ventricle through the torn aortic valve. Compare with competent aortic valve in Fig. 4.

In this series of dogs with chronic mitral insufficiency, the left ventricular punctures were made without fluoroscopic control and occasionally two or three attempts were required to enter the left ventricle. In a subsequent group of dogs, fluoroscopic control was used and this appears to be a useful aid in the correct positioning of the needle during direct puncture of the left ventricle. By injecting a small a small dose of Urokon under fluoroscopic observation, the position of the needle may be more precisely determined.

Electrocardiograms taken at the time of the direct puncture, and during the passage of the catheter through the aortic valve, have revealed frequent



A. B

Fig 12.—Complications of direct left ventricular puncture and dye injection A, Intramyocardial extravasation of dye; B, accidental entrance of needle into coronary artery

premature ventricular contractions. In the series of 17 injections of contrast medium in the ascending aorta, there was noted only transient effect on the electrocardiogram and no mortality.

## COMMENTS

There appears to be little doubt that the normal mitral and aortic valves prevent regurgitation of injected radiopaque material during regular sinus rhythm. When these valves are rendered insufficient by the techniques used in this study, there has been no difficulty in recording either mitral or aortic reflux on a single roentgenogram. These experimental valvular lesions, however, differ from the lesions in the diseased valves of the human heart. Mitral insufficiency created in the dog by rupturing a valve leaflet is quite different from the lesion which results in man from the processes of shortening and thickening of the leaflets, or by dilatation of the mitral annulus. Aortic insufficiency experimentally produced in the dog by perforating an otherwise normal cusp has little anatomic correspondence to an incompetent aortic valve due to the scarring of the cusps and separation of the commissures. These important anatomic differences between the naturally occurring and the experimentally created valvular lesion may have corresponding functional variations. However, the essential feature of free retrograde reflux through the incompetent valve has been duplicated experimentally. It is this reflux which is responsible for the major hemodynamic change in mitral or aortic insufficiency and it appears clear that cardioangiographic technique detects its presence.

Quantitation of the degree of regurgitation is a much more difficult problem than the mere demonstration of its presence. Alterations in rate and contractility of the ventricle, changes in peripheral arterial pressure variations in the speed of injection of the radiopaque medium, changes in roentgenographic technique, and transient arrhythmias at the time of injection, all may distinctly vary the density of the shadow of the regurgitating dye. Rapid dilution of the jet of dye in a large chamber may make the reflux appear smaller than an equivalent jet in a smaller cardiac chamber. A rise in arterial pressure head favors mitral reflux, while a transient episode of hypotension during the procedure could reduce the degree of regurgitation. Analysis of double outlet chambers has shown how significantly the speed of ventricular contraction may influence the degree of mitral reflux.[19] Any attempt at densitometric quantitation of the insufficiency must take into account these many factors. However, as can be seen from the illustrations (Figs. 6 and 7), large insufficiencies seem to give a denser opacification of the left atrium than do small ones, and there can be no doubt that the increasing size of the cardiac chambers is well demonstrated with this technique.

It is apparent from Figs. 5 and 9 that mitral insufficiency can be detected by this method when this valvular lesion exists alone or when it is associated with mitral stenosis. The stenotic valve actually prolongs circulation time through the left auricle and facilitates roentgen visualization of this chamber when mitral insufficiency and stenosis are combined. Since small regurgitant jets may not be a contraindication to surgery in mitral stenosis, quantitation of the amount of reflux becomes an important consideration. As previously stated, it is our impression that a dynamically significant insufficiency usually gives a denser opacification of the left atrium than does a minor jet. In the series of radiographs in Fig. 7, the reduction in the size of a regurgitant jet is illustrated. The spot film technique, when adequately carried out with rapid injection and proper timing of the radiographic exposure, has not failed to demonstrate significant mitral insufficiency when present.

The transference of cardioangiographic technique for the diagnosis of mitral or aortic insufficiency to man would appear to depend primarily on the question of the safety of the procedure. Björk[16] described severe reactions after left atrial injection of dye. The two dogs that died in this study, and the patient in the report of Smith[8] who developed ventricular fibrillation, were all studied by the direct puncture technique. The major hazard of this needle technique would appear to be the inadvertent injection of the medium into the myocardium (Fig. 12, *A*), or laceration of a coronary vessel (Fig. 12, *B*). Neither of these complications can occur with retrograde arterial catheterization of the left ventricle. Accidental entrance of the retrograde catheter into a coronary orifice in man is a distinct possibility, but with careful fluoroscopy and attention to the electrocardiogram this should be avoidable. Fluoroscopic control will help avoid the hazard of passage of the catheter into a carotid artery and delivery of a large dose of contrast material to the brain. Injury to the aortic valve in passing the catheter into the ventricle may be avoided by gentle manipulation under fluoroscopy and by providing the retrograde catheter with a sharply curved tip. Arrhythmias encountered due to the introduction of the catheter into the aorta and left heart should be no greater than those met with in the right heart catheterization. However, it would appear that because of greater stability of the heart in the well-ventilated patient, the procedure should be carried out with an anesthetist in attendance, administration of 100 per cent oxygen just prior to the dye injection, and continued adequate oxygenation after the procedure.

Some of these conceivable complications are less likely to occur with dye injections through a catheter in the aorta for the demonstration of aortic insufficiency. In the present study, this type of injection was carried out without noticeable complication. It must be remembered that a virtually undiluted bolus of dye enters the coronary arteries during aortic injection and with preexisting coronary insufficiency this type of aortography has distinct potential hazards. Repeated and closely spaced contrast medium injections should be avoided. The problem of circulatory embarrassment to the limb in which the arterial catheterization is performed will be a real one unless careful preoperative assessment of collateral circulation and meticulous technique is observed. The radial artery has been used routinely in a large number of cases by Jönsson, Brodén, and Karnell.[20] In adults, as shown by the experience of Peirce,[21] femoral catheterization with small catheters can be performed without danger to the limb.

## SUMMARY

1 In dogs, the normal mitral and aortic valves prevent retrograde reflux of radiopaque contrast medium

2 Mitral insufficiency is readily demonstrated on roentgenograms by the injection of contrast medium into the left ventricle, since the incompetent mitral valve permits reflux of dye into the left atrium

3 Aortic insufficiency can be detected when contrast material, injected through a catheter in the ascending aorta, regurgitates into the left ventricle

4 The combination of mitral stenosis and insufficiency is radiologically demonstrable by the injection of contrast material into the left ventricle

5 The injection of contrast material into the left ventricle through the retrograde aortic catheter appears to be safer than the direct puncture technique

The authors wish to thank Mrs Audrey Raisbeck for her expert technical assistance in the laboratory

## REFERENCES

1 Gordon, A J, Kuhn, L, Amram, S S, Donoso, E, and Braunwald E  Left Atrial "Pulmonary Capillary" and Esophageal Balloon Pressure Tracings in Mitral Valve Disease, Brit Heart J (In Press)
2 Wynn, A, Mathews, M B, McMillan, I R R, and Daley, R  The Left Auricular Pressure Pulse in Normals and in Mitral Disease, Lancet 263 216 1952
3 Reboul, H, and Racine, M  Ventriculographie cardiaque expérimentale, Presse méd 37. 763, 1933
4 Nuvoli, I  Arteriographia dell' Aorta Toracica Mediante Puntura dell' Aorta Ascendente o del Ventricolo S, Policlinico 43 227, 1936
5 Radner, S  An Attempt at the Roentgenologic Visualization of Coronary Blood Vessels in Man, Acta radiol 26 497, 1945
6 Ponsdomenech, E R, and Nuñez, V B  Heart Puncture in Man, Am Heart J 41 643, 1951
7 Nuñez, V B, and Ponsdomenech, E R  Heart Puncture II Cardioangiography, Clinical and Electrocardiographic Results, Am Heart J 41 855, 1951
8 Smith, P W, Wilson, C W, Gregg, H A, and Klassen, K P  Cardioangiography, J Thoracic Surg 28 273, 1954
9 Smith, P W, Gregg, H A, and Klassen, K P  The Diagnosis of Mitral Regurgitation by Cardioangiography, Circulation 12 777, 1955
10 Castellanos, A, and Pereiras, R  Counter Current Aortography, Rev cubana cardiol 2 187, 1948
11 Radner, S  Thoracal Aortography by Catheterization From the Radial Artery, Acta radiol 29 178, 1948
12 Jonsson, G, Broden, B, Hanson, H E, and Karnell, J  Visualization of Patent Ductus Arteriosus Botalli by Means of Thoracic Aortography, Acta radiol 30 81, 1948
13 Stephens, H B  Discussion of Bradshaw, O'Neill, and Hightower  J Thoracic Surg 17. 210, 1948
14 Jonsson, G  Thoracic Aortography by Means of a Cannula Inserted Percutaneously Into the Carotid Artery, Acta radiol 31 376, 1949
15 Katz, L N, Malinow, M R, Kondo, B Feldman, D and Grossman, N  The Volume Elasticity of the Aorta in the Intact Dog, Am Heart J 33 319, 1947
16 Bjork, V O, Kjellberg, S R, Malmstrom, G, and Rudhe, U  The Diagnosis of Mitral Insufficiency, Am Heart J 49 719, 1955
17 Kjellberg, S R, Mannheimer, E, Rudhe, U, and Jonsson, B  Diagnosis of Congenital Heart Disease, Chicago, 1955 Year Book Publishers, Inc
18 Boorema, I, and Blickman, J R  Reduced Intrathoracic Circulation as an Aid in Angiocardiography, J Thoracic Surg 30 129, 1955
19 Rodbard, S, and Williams, F  The Dynamics of Mitral Insufficiency, Am Heart J 48 521, 1954
20 Jonsson, G, Brodén, B, and Karnell, J  Thoracic Aortography, Acta radiol (suppl) 89, 1951
21 Peirce, C E  Recent Advances in Radiological Studies of the Vascular System, Maryland State Med J 2 635, 1953

jets may not be a contraindication to surgery in mitral stenosis, quantitation of the amount of reflux becomes an important consideration. As previously stated, it is our impression that a dynamically significant insufficiency usually gives a denser opacification of the left atrium than does a minor jet. In the series of radiographs in Fig. 7, the reduction in the size of a regurgitant jet is illustrated. The spot film technique, when adequately carried out with rapid injection and proper timing of the radiographic exposure, has not failed to demonstrate significant mitral insufficiency when present.

The transference of cardioangiographic technique for the diagnosis of mitral or aortic insufficiency to man would appear to depend primarily on the question of the safety of the procedure. Björk[16] described severe reactions after left atrial injection of dye. The two dogs that died in this study, and the patient in the report of Smith[8] who developed ventricular fibrillation, were all studied by the direct puncture technique. The major hazard of this needle technique would appear to be the inadvertent injection of the medium into the myocardium (Fig. 12, *A*), or laceration of a coronary vessel (Fig. 12, *B*). Neither of these complications can occur with retrograde arterial catheterization of the left ventricle. Accidental entrance of the retrograde catheter into a coronary orifice in man is a distinct possibility, but with careful fluoroscopy and attention to the electrocardiogram this should be avoidable. Fluoroscopic control will help avoid the hazard of passage of the catheter into a carotid artery and delivery of a large dose of contrast material to the brain. Injury to the aortic valve in passing the catheter into the ventricle may be avoided by gentle manipulation under fluoroscopy and by providing the retrograde catheter with a sharply curved tip. Arrhythmias encountered due to the introduction of the catheter into the aorta and left heart should be no greater than those met with in the right heart catheterization. However, it would appear that because of greater stability of the heart in the well-ventilated patient, the procedure should be carried out with an anesthetist in attendance, administration of 100 per cent oxygen just prior to the dye injection, and continued adequate oxygenation after the procedure.

Some of these conceivable complications are less likely to occur with dye injections through a catheter in the aorta for the demonstration of aortic insufficiency. In the present study, this type of injection was carried out without noticeable complication. It must be remembered that a virtually undiluted bolus of dye enters the coronary arteries during aortic injection and with preexisting coronary insufficiency this type of aortography has distinct potential hazards. Repeated and closely spaced contrast medium injections should be avoided. The problem of circulatory embarrassment to the limb in which the arterial catheterization is performed will be a real one unless careful preoperative assessment of collateral circulation and meticulous technique is observed. The radial artery has been used routinely in a large number of cases by Jönsson, Brodén, and Karnell.[20] In adults, as shown by the experience of Peirce,[21] femoral catheterization with small catheters can be performed without danger to the limb.

## SUMMARY

1. In dogs, the normal mitral and aortic valves prevent retrograde reflux of radiopaque contrast medium.

2. Mitral insufficiency is readily demonstrated on roentgenograms by the injection of contrast medium into the left ventricle, since the incompetent mitral valve permits reflux of dye into the left atrium.

3. Aortic insufficiency can be detected when contrast material, injected through a catheter in the ascending aorta, regurgitates into the left ventricle.

4. The combination of mitral stenosis and insufficiency is radiologically demonstrable by the injection of contrast material into the left ventricle.

5. The injection of contrast material into the left ventricle through the retrograde aortic catheter appears to be safer than the direct puncture technique.

The authors wish to thank Mrs. Audrey Raisbeck for her expert technical assistance in the laboratory.

## REFERENCES

1. Gordon, A. J., Kuhn, L., Amram, S. S., Donoso, E., and Braunwald, E.: Left Atrial, "Pulmonary Capillary" and Esophageal Balloon Pressure Tracings in Mitral Valve Disease, Brit. Heart J. (In Press).
2. Wynn, A., Mathews, M. B., McMillan, I. K. R., and Daley, R.: The Left Auricular Pressure Pulse in Normals and in Mitral Disease, Lancet 263: 216, 1952.
3. Reboul, H., and Racine, M.: Ventriculographie cardiaque experimentale, Presse méd. 37: 763, 1933.
4. Nuvoli, I.: Arteriographia dell' Aorta Toracica Mediante Punctura dell' Aorta Ascendente o del Ventricolo S., Policlinico 43: 227, 1936.
5. Radner, S.: An Attempt at the Roentgenologic Visualization of Coronary Blood Vessels in Man, Acta radiol. 26: 497, 1945.
6. Ponsdomenech, E. R., and Nuñez, V. B.: Heart Puncture in Man, Am. Heart J. 41: 643, 1951.
7. Nuñez, V. B., and Ponsdomenech, E. R.: Heart Puncture: II. Cardioangiography, Clinical and Electrocardiographic Results, Am. Heart J. 41: 855, 1951.
8. Smith, P. W., Wilson, C. W., Gregg, H. A., and Klassen, K. P.: Cardioangiography, J. Thoracic Surg. 28: 273, 1954.
9. Smith, P. W., Gregg, H. A., and Klassen, K. P.: The Diagnosis of Mitral Regurgitation by Cardioangiography, Circulation 12: 777, 1955.
10. Castellanos, A., and Pereiras, R.: Counter Current Aortography, Rev. cubana cardiol. 2: 187, 1948.
11. Radner, S.: Thoracal Aortography by Catheterization From the Radial Artery, Acta radiol. 29: 178, 1948.
12. Jönsson, G., Broden, B., Hanson, H. E., and Karnell, J.: Visualization of Patent Ductus Arteriosus Botalli by Means of Thoracic Aortography, Acta radiol. 30: 81, 1948.
13. Stephens, H. B.: Discussion of Bradshaw, O'Neill, and Hightower: J. Thoracic Surg. 17: 210, 1948.
14. Jönsson, G.: Thoracic Aortography by Means of a Cannula Inserted Percutaneously Into the Carotid Artery, Acta radiol. 31: 376, 1949.
15. Katz, L. N., Malicow, M. R., Kondo, B., Feldman, D., and Grossman, N.: The Volume Elasticity of the Aorta in the Intact Dog, Am. Heart J. 33: 319, 1947.
16. Björk, V. O., Kjellberg, S. R., Malmstrom, G., and Rudhe, U.: The Diagnosis of Mitral Insufficiency, Am. Heart J. 49: 719, 1955.
17. Kjellberg, S. R., Mannheimer, E., Rudhe, U., and Jönsson, B.: Diagnosis of Congenital Heart Disease, Chicago, 1955, Year Book Publishers, Inc.
18. Boerema, I., and Blickman, J. R.: Reduced Intrathoracic Circulation as an Aid in Angiocardiography, J. Thoracic Surg. 30: 129, 1955.
19. Rodbard, S., and Williams, F.: The Dynamics of Mitral Insufficiency, Am. Heart J. 48: 521, 1954.
20. Jönsson, G., Brodén, B., and Karnell, J.: Thoracic Aortography, Acta radiol. (suppl.) 89, 1951.
21. Peirce, C. E.: Recent Advances in Radiological Studies of the Vascular System, Maryland State Med. J. 2: 635, 1953.

# LUNG DENERVATION[*] IN THE THERAPY OF INTRACTABLE BRONCHIAL ASTHMA

DANIEL DIMITROV-SZOKODI, M.D., ALEXANDER HUSVÉTI, M.D., AND
GYULA BALOGH, M.D.
BUDAPEST, HUNGARY

THE problem of the therapy of intractable bronchial asthma is well known. The use of ACTH, cortisone, and various antibiotics has meant a great advance when one considers the difficulty of attaining good results with the older drug therapy. However, after studying the earlier literature, it became clear that with the older treatment the serious condition of a number of asthmatic patients precluded anything more than symptomatic therapy. Following the asthmatic secondary changes, the patient's working ability steadily decreases, he becomes unable to work, and the prognosis is poor.

In order to treat bronchial asthma that resisted internal treatment, the idea of attacking the vegetative nervous system was tried long ago. First, in 1923, Kümmel obtained results with cervicothoracic sympathectomy, and, in 1924, Kappis with vagotomy on the right side. By the addition to the anatomic and pathophysiologic knowledge and the development of better techniques in thoracic surgery during the last three decades, several authors have dealt with the surgical treatment of intractable bronchial asthma and have tried to perfect the attack on the vegetative nervous system (Phillips, Scott, Leriche and Fontaine, Rienhoff and Gay, Miscall and Rovenstien, Carr and Chandler, Blades, Klassen, Abbott, and others).

In the Sebestény Clinic, after having tried and failed with every conservative treatment in a patient, we have then considered the possibility of surgical intervention in cases of intractable bronchial asthma as an "ultimum refugium" (Dimitrov-Szokodi).

The advisability and efficiency of the surgical treatment of bronchial asthma are still matters for discussion. In the literature, data concerning the theoretical basis of the various surgical procedures are also very contradictory. Therefore, before reaching a practical solution, it was essential to consider a few questions.

1. What is the role of the lung's extrinsic vegetative fibers in the pathology of bronchial asthma?

2. What kind of vegetative intervention seems to be anatomically and pathophysiologically best founded?

---

From the III. Surgical Clinic, University, Budapest (Director: Prof. J. Kudász).

Received for publication Dec. 20, 1955.

[*] We mean by this, the bilateral intervention of extrinsic vagus and sympathetic nerves of the lung.

3. Can the "nil nocere" principle be followed when operating on the vegetative fibers of the lung?

1. In the pathogenesis of bronchial asthma, besides the ordinary features, we wish to emphasize the important role of the lung's allergic infectious processes (Hajós and Hetényi, among others).

The question is, how can these processes lead to asthmatic attack? For the understanding of the pathomechanism of asthmatic attack we refer to Danielopolu's theory. According to this theory, the often recurring stimuli of the allergic and infectious processes (the release of "H materials") taking place in the lung, lead to the excitation of the vegetative centers via afferent pathways. This stimulation is transferred to the lung by efferent pathways and it further increases the changes and damages. The developing bronchial spasm, swelling of the mucous membrane, hypersecretion, stasis of the pulmonary vascular system ([Tsuji] parasympathetic effects) create unfavorable conditions, the inhibition of expectoration leads to retention of the secretion, and to increase a tendency for infections also. The mentioned changes caused by the efferent impulse, i.e., those chemical mediators (acetylcholine, histamine, etc.), released in the meantime, stimulate again the already more sensitive receptors. There is a vicious circle of reflex actions taking place between the vegetative centers and the lung. The extrinsic vegetative pathways of the lung play an important role in the communication of this reflex. After the development of this vicious circle, not only can the original cause lead to asthmatic attack, but a wide scale of other mild and shortlasting extero- and interoceptive stimuli (psychic, neural, humoral stimuli) also can produce a continuous vicious circle increase, which can be manifested in a longer asthmatic paroxysm.

On the basis of the research of Pavlov and his school, there is a possibility of developing conditioned reflexes owing to the frequent repetition of the attacks. The importance of conditioned reflexes in the asthmatic condition is supported also by our own clinical observations.

The pathologic role of the vegetative nervous system in asthma, caused either by allergic condition or by infection, becomes manifest also in numerous experimental studies. The experiments of Reitler, Friedberger, Bogendorfer, Moser and Tusch, Kiszely and Migma, Filipp, Szentiványi and Mess, and others, proved that with the experimental lesion of the central parts of the vegetative nervous system (brain stem, tuber cinereum, etc.) the development of general anaphylactic allergic signs can be prevented. On the other hand, the rise of local anaphylactic allergic signs can be inhibited or greatly reduced in their intensity by the interruption of the peripheral vegetative innervation (Buchwald, Wyropajev, Keztyus and his co-workers, among others).

Similar experimental results are obtained also with the prevention or reduction in intensity of the development of local infectious processes by the interruption of peripheral fibers (Szperanszkij, Mansfeld).

2. Our aim was to eliminate the pathologic role, just outlined, of the vegetative nervous system by surgical intervention in the case of intractable

bronchial asthma. It is necessary to give a short review of the lung's innervation for the determination of the suitable spot and extent of the operation.

The lung is innervated by the vagus and the sympathetic nerves, both of which contain afferent (sensory) and efferent (motor) fibers. From the vagus nerve the pulmonary branches arise on the portion extending from the origin of the recurrent nerve to the pulmonary ligament. The main sympathetic supply of the lung comes from fibers from the ganglia of the second to fifth thoracic vertebrae of the sympathetic chain (Braeucker, Kuntz, Kiss, Mitchell, Dijkstra). The vagus and the sympathetic nerves have an extensive anastomosis with each other and also with the contralateral similar fibers. Therefore, both lungs receive vagus and sympathetic fibers from the same as well as from the contralateral side. Thus, the lung's innervation is ''bilateral and bisystematic'' (White and Smithwick).

From the physiologic point of view, the vagus nerve cannot be considered to have only cholinergic effect and the innervation coming from the sympathetic chain to have only adrenergic effect (De Burgh Daly, von Euler, Levin, Hebb, and others). For example, bronchial asthma can be produced also with the stimulation of the sympathetic chain. According to Ferenc Kiss, the fibers having an antagonist function cannot be localized to certain nerves, but are mixed.

In order to work out a better surgical method, we have reviewed the experiences in the operations performed up to the present time. From them it can be concluded that the success of the operation is proportional to the degree of denervation. After having accomplished radical bilateral denervation (Rienhoff and Gay), lasting good results—eight years—have been observed. According to the data in the literature, if only the vagus nerve or the sympathetic denervation is completed, the remaining system is still able to maintain asthmatic conditions. These experiences can be understood from the described anatomic and physiologic conditions.

Considering these aspects, it seemed logical to perform an intervention which extended to both sides equally and which involved, in one intrathoracic operation, the interruption of the pulmonary branches of the vagus—from the recurrent nerve to the pulmonary ligament (Klassen, Clark)—and the removal of the lung's main sympathetic control transmitted by the ganglia of the second to fifth thoracic vertebrae (Miscall and Rovenstine, Carr and Chandler). Hence, a complete sympathectomy can be produced, and at the same time, the dangerous operations carried out in the hilus can be avoided, although the results are good (Rienhoff and Gay, Blades and his co-workers). These authors, besides transecting the pulmonary plexus, denude the main bronchi and the great pulmonary arteries and veins to attain a complete sympathetic denervation.

3. Prior to the application to the vegetative nervous system of the operation based on the knowledge mentioned above, the question arose, will any damage of the respiratory function follow?

Fontaine and Herrmann proved with experiments on animals that, after the transection of the pulmonary branches of the vagus nerve and the upper thoracic sympathectomy, the function of respiration and circulation did not change, and no kind of defect and damage could be observed. The studies of bilateral radical intervention on man prove, also, that after the operation, the respiration, cough, and expectoration are undisturbed. Neurohistologic examinations, after the interruption of the lung's extrinsic vegetative pathways, show a relative automatism to the intrapulmonary (intrinsic) nervous system (Dijsktra, among others). Therefore, after the bilateral denervation of the lung, the remaining part of the neurohormonal regulating system, restoration of a stabilized condition of the thorax, and intact dynamic factors can further ensure—without noticeable defect—the function of the lung and the adaptability of the respiration.

*Our Examinations Before the Operation Referring to the Pathomechanism of the Asthmatic Attack and the Expected Result of the Surgical Treatment.*—

In patients having intractable bronchial asthma, we have produced attacks before the operation by giving histamine (Figs. 1, *C* and 3, *C*) and have registered the respiratory function with a spirograph. In 8 patients, we have obtained an average of 25 per cent reduction in the vital capacity and a 16 per cent reduction in the pulmonary ventilation rate maximum. These asthmatic signs can be compared with the original asthmatic attack on the basis of the reduced respiratory function as well as the clinical picture (Dzsinich) (Figs. 1, *D* and 3, *D*). In 8 patients, the vital capacity was reduced on an average of 45 per cent and the pulmonary ventilation rate maximum 46 per cent during an attack. We suppose that in these cases, not only the local pharmacologic effect of the histamine appears to function, but the histamine as an initiating stimulus starts the vicious circle leading to attack.

Since both the sympathetic and the vagus nerves participate in the transmitting of the afferent and efferent impulses maintaining the vicious circle, we have attempted the relief of the genuine attack as well as of the attack caused by histamine by the temporary elimination of the conduction of these pathways. We have blocked the vagosympathetic pathway system in th neck and paravertebrally in the upper thoracic section with Novocain (Visnyevszkij). At each place we have injected 20 to 30 c.c. of ½ per cent Novocain. After the blockade, the attacks have ceased (Figs. 1, *E* and 3, *E*). It is therefore possible that the reflex action maintaining the attacks has been interrupted (the vicious circle of Danielopolu). We have been able to demonstrate objectively the relaxation of the attack after the Novocain blockade with a spirographic curve record. Successful, temporary cessation of the asthmatic attack with Novocain blockade has proved the important role of the lung's extrinsic pathways in the asthmatic attack.

We have been able to draw conclusions concerning the degree of the expected success of the operation from the effects of the vagosympathetic blockade used as a test before the intervention. We also gained information about the reversible or irreversible character of the secondary changes accompan

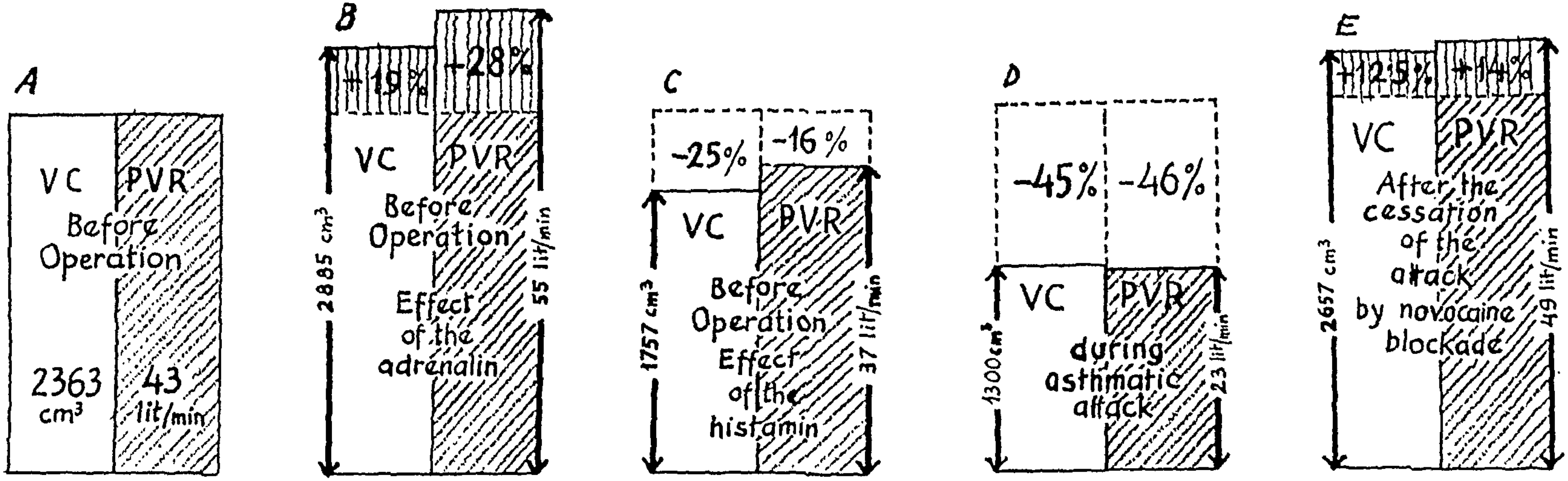


Fig. 1.—The average values of the respiratory function of 8 patients examined before denervation. VC = vital capacity. PVRmax = pulmonary ventilation rate maximum.

the asthmatic condition (emphysema, among others) with a spirographic examination after administering adrenalin to relax the latent bronchial spasm during the period free of attack. (Figs 1, *A*, *B* and 3, *A*, *B*). (In this period, the vital capacity is 2,363 c.c., the pulmonary ventilation rate maximum is 55 L. per minute. After giving adrenalin, the vital capacity is 2,885 c.c. [an increase of 19 per cent], and the pulmonary ventilation rate maximum is 55 L. per minute [a 28 per cent increase]*). If the attack has ceased after the blockade and the respiratory function values have increased as an effect of the adrenalin given in the period free of attack, we have considered intervention to be justified and assumed that we could expect good results.

Up to the present time we have operated on 30 patients with our surgical procedure, but in this article we are dealing with 19 patients only in summarized evaluation, because we consider in these patients only that the time of observation is sufficient.

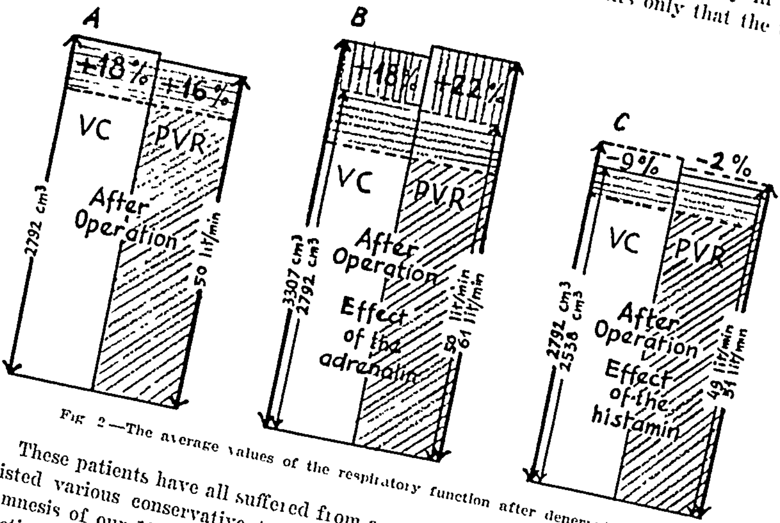


Fig 2—The average values of the respiratory function after denervation

These patients have all suffered from a serious asthmatic status which has resisted various conservative treatments. We want to emphasize from the anamnesis of our 19 patients that 16 of them had some lesion (in most cases infectious) of the respiratory system preceding the development of bronchial asthma, 2 patients suffered damage of the central nervous system (commotio cerebri), and one patient had a psychosis after childbirth. Every one of these coincided with the beginning of the bronchial asthma.

We could not determine the specific stability of the allergens. However, in several cases, due to the occupation of the patients (e.g., druggist, textile

---

*Average values of the respiratory function.

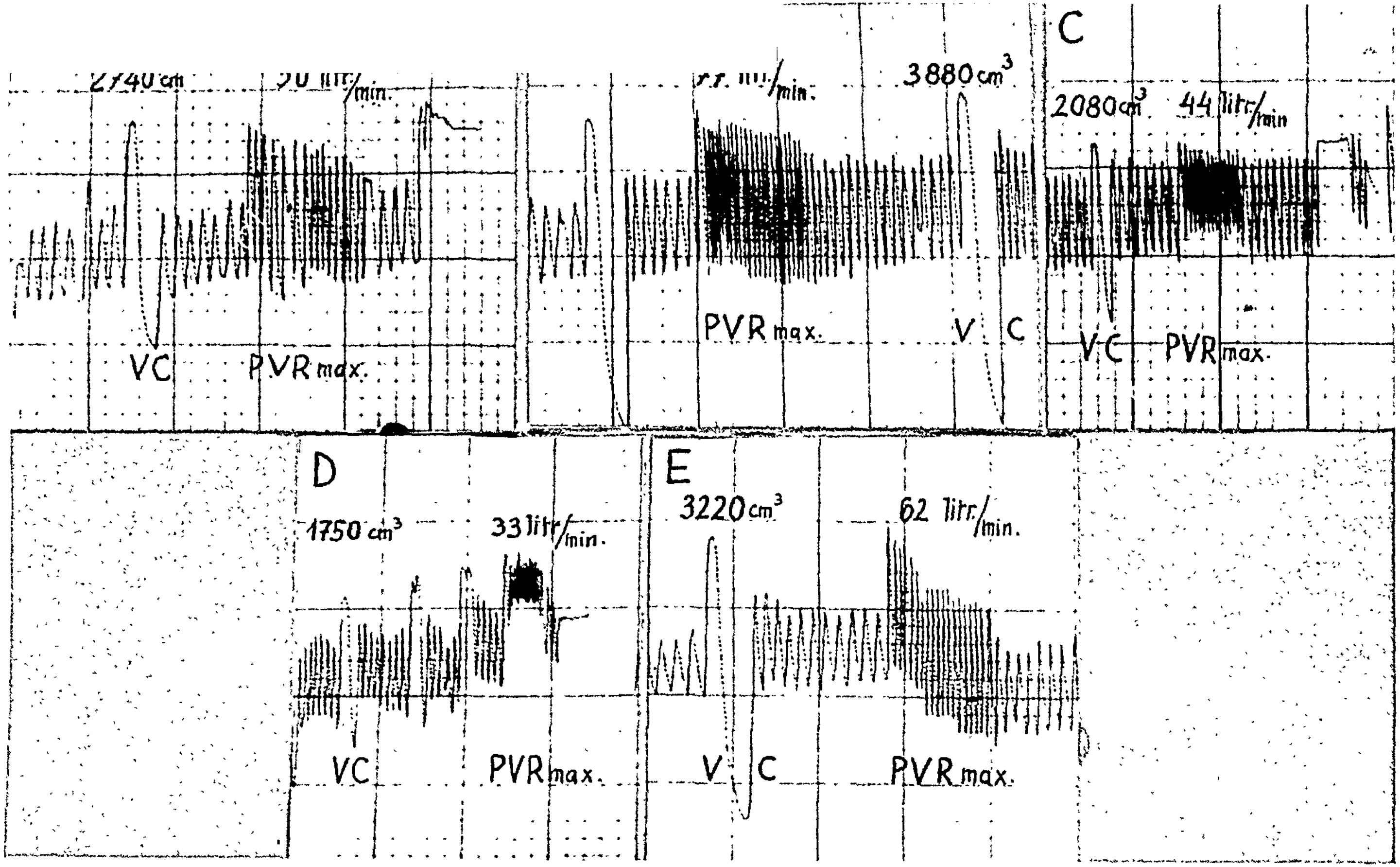


Fig. 3.—The spirographic curves of Patient T. P. before denervation. VC = vital capacity. PVRmax = pulmonary ventilation rate maximum; 1 ☐ = 200 c.c.

*A*, In the period free of attack, the respiratory function decreased. The ideal VC for this patient would be 4,480 c.c., the PVRmax would be 72 L./min.

*B*, The effect of 2 mg. of adrenalin given in the period free of attack. The VC and the PVRmax were considerably increased. The expiration was especially improved.

*C*, After giving 0.5 mg. histamine, the respiration became dyspneic and the frequency increased. Owing to the reduction of the VC and the PVRmax, it resembles the curve taken during the attack.

*D*, The curve taken during the asthmatic attack is characteristic; little reserve air, during the definition of the PVRmax the patient ventilates with complementer air.

*E*, After the bilateral vagosympathetic blockade carried out during the attack, the VC and the PVRmax greatly increased above the values found in the period free of attack.

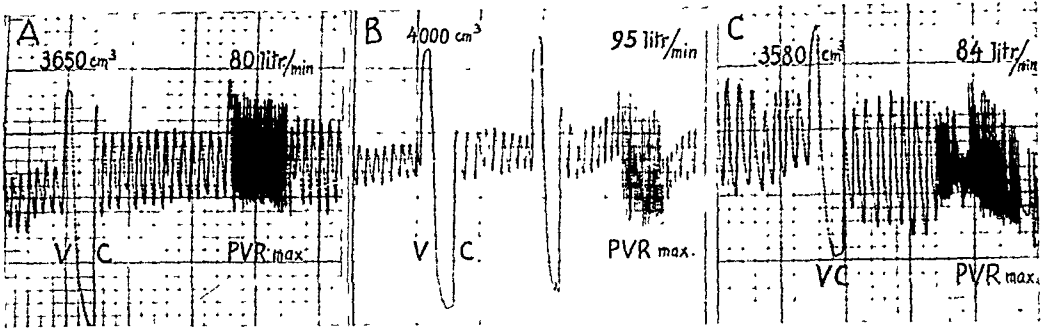


Fig 4.—Spirographic curves taken three months after the bilateral denervation. *A*, Marked increase in the VC and PVR-max after operation; reserve air increased. *B*, After giving ½ mg of adrenalin, the VC and PVRmax show the highest value among all other curves (increased adrenalin sensitivity). *C*, After giving 0.5 mg. histamine, the respiratory function did not change considerably (paroxysmal syndrome did not appear)

and leatherworker) a possible sensitivity to certain materials could be assumed. A large number of the patients had previously gone through various nonspecific desensitivity processes without success.

Of the 19 patients, there were 12 men and 7 women. Their ages were between 19 and 55, most of them were between 40 and 50 years of age. The period of their illness was between 5 and 20 years; most of our patients had suffered from asthma for more than 10 years.

The examination of our patients covered particularly the condition of the cardiorespiratory system. We considered as contraindications to surgical treatment only those conditions, which would prevent a major surgical operation (decompensation, among others).

In preparation for intervention, we considered it important to assure a period free of attack, and instituted the removal of the bronchial secretion, adequate treatment of the infectious processes with antibiotics, oxygenization, cardiac reinforcement, and other measures.

*The Essential Part of the Surgical Technique**.—Bilateral intrathoracic intervention (in the fourth intercostal space on the right side, and in the fifth intercostal space on the left side) is made with a parascapular incision, using local anesthesia. After opening the mediastinal pleura, we resect the branches of the vagus nerve going to the lung from the recurrent nerve to the pulmonary ligament. Lifting out the vagus trunk from the mediastinum, we suture the mediastinal pleura underneath in order to prevent regeneration. After the opening of the parietal pleura, we remove the sympathetic ganglion chain of the second to fifth thoracic vertebrae by the transection of the rami communicantes. We tested starting the denervation first on the right, then on the left side, but realized that the order of succession did not make any difference. We performed the contralateral operation after the complete postoperative restitution following the first operation.

We have accomplished bilateral lung denervation in 12 patients, and unilateral lung denervation in 7 patients. There was one death directly after the second intervention during the bronchoscopic aspiration. One of our patients died 9 months after the unilateral denervation with bilateral pneumonia complicated with epidemic influenza. According to the postoperative symptoms, this death could not have been due to the operation.

*Surgical Complications.*—The most critical was a 3-minute cardiorespiratory syncope following intrathoracic Novocain blockade of the vagus trunk which we have discontinued, using the ordinary methods of resuscitation. Technical errors due to inexperience have caused the only recurrent nerve paresis observed and, in one case, a temporary atony of the stomach followed strong stretching of the vagus nerve. In 2 cases we have observed a Horner syndrome lasting for a few months. A spontaneous pneumothorax occurred in one case in the postoperative period that was associated with the great emphysema of the asthmatic patients.

---

*Detailed description in J. Internat. de Chir. Bruxelles, 13: 588, 1953.

The success of our surgical treatment has been evaluated on the results of the data of the objective examinations, and of the changes noted in the condition of the patients

*Results of the Objective Examinations*—

*Changes in the respiratory system* We have determined with bronchoscopic examination that the changes in the mucous membrane have regressed The mucous membrane was swollen, mucilaginous, or inflamed before operation in 12 patients Findings in 5 of these patients became negative after operation In 5 patients, the changes of the mucous membrane have been relieved In one patient who has had only unilateral denervation so far, no change has followed Parallel with the changes of the mucous membrane, expectoration has ceased or decreased Before operation, 11 patients had a large quantity of sputum After operation, in 8 of them the production has practically discontinued There has been a decrease in 2, and an increase in one The purulent character of the sputum in 6 patients has disappeared In 2 of these cases, we have performed lobectomy because of bronchiectasis In the sputum of 15 patients we have found eosinophils After operation, this eosinophilia has disappeared in 12 patients, in the other 3, it has decreased Thus, we have established that on the operated side the allergic and infectious processes of the bronchial system have either stopped or been relieved

With physical examination it has became manifest that after the surgical treatment a favorable change has occurred in the inspiratory position of the lung, if rigidity of the thorax has not developed up to this time The characteristic asthmatic auscultatory signs have ceased or considerably decreased depending on whether uni or bilateral denervation was performed, that is, what was the degree of reversibility of the changes before the operation?

*Radiographic morphologic examination* We have showed that after the denervation there is improvement or normalization of the secondary changes accompanying the asthmatic condition, especially as demonstrated by indirect signs of improvement of emphysema (in the horizontal position of the ribs, in the deep position of the diaphragm) (Fig 5) Among the 11 bilaterally denervated patients, improvement was observed in 7 cases, among the 7 unilaterally denervated patients, 3 have improved The character of the emphysema in these patients has been reversible and functional up to now In 3 patients, among the bilaterally denervated, the changes have been irreversible In one case, however, we have not observed any improvement, owing to the surgical complication of recurrent nerve paresis

It has been well demonstrated by bronchograms that the increased tone of the bronchi, compared with the picture before the intervention, shows that the diffuse spastic condition has disappeared or been reduced according to whether the operation has been done uni or bilaterally (Fig 6) Relaxation in the spastic condition of the bronchi on the operated side could be observed even after the unilateral denervation In 4 cases, we have showed the destructive changes of the bronchi before the surgical [illegible] tasis

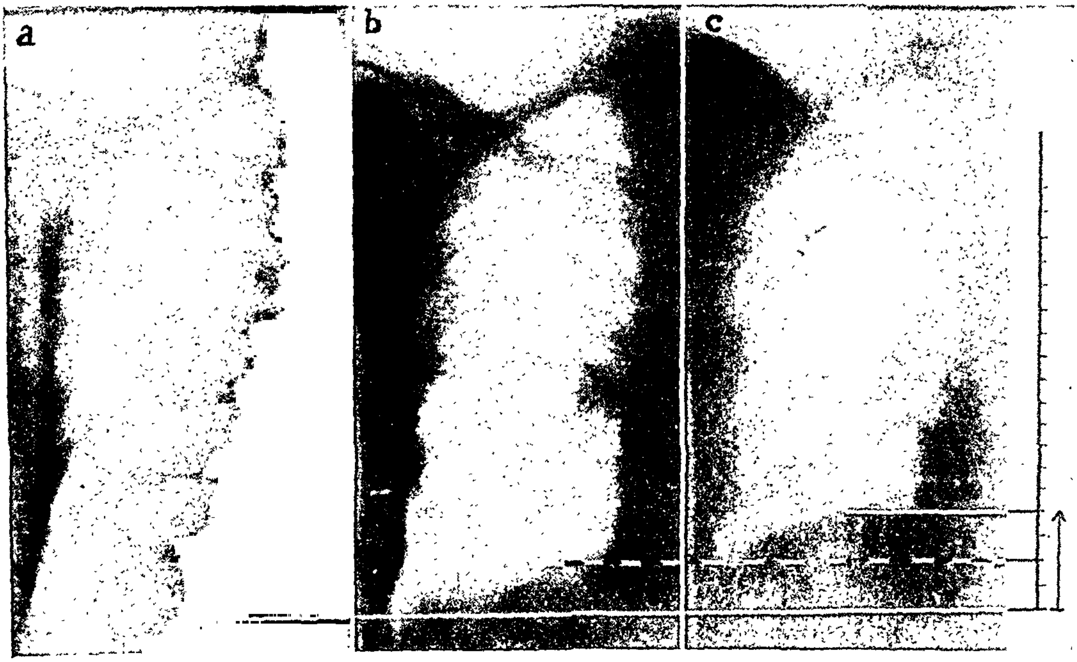


Fig. 5.—X-rays taken from the right side of the thorax of K. L. which show the changes that occurred in the position of the diaphragm. *a*, The right diaphragm stands in the level of the eleventh rib before denervation. *b*, One month after denervation the right diaphragm stands a little under the tenth rib. *c*, Fourteen months after denervation the right diaphragm stands in the line of the tenth rib.

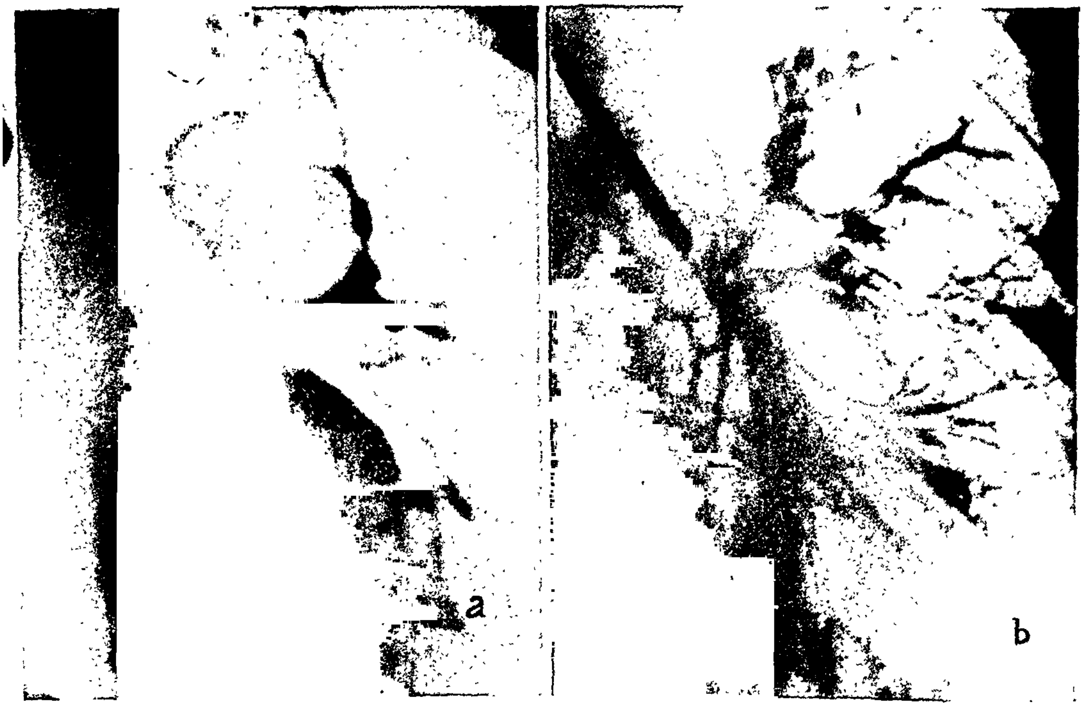


Fig. 6.—A comparison of the bronchograms of T. P. taken before and after the denervation; *a*, spastic condition in the whole bronchial system before denervation. In spite of the pressure of the opaque medium, the peripheral bronchi did not fill up. *b*, After denervation the spastic signs disappeared and the peripheral branches easily filled up. To a certain extent, alveolar filling can also be observed.

in 2 cases when we have performed lobectomy at the same time as denervation, and deforming bronchitis in the other 2 cases, which remained unchanged after the operation.

On examining the function of the bronchi of asthmatic patients by means of bronchography, we have found that after denervation the bronchi approached a normal condition during coughing except in instances of a deforming bronchitis.

*Examination of the respiratory function.* Owing to the constant bronchial spasm and the reversible or irreversible emphysema existing in the asthmatic condition, the efficiency and the adaptability of the lung in the period free of attack is changed also. We have determined the reduced respiratory function before the operation and the improvement after the operation by means of objective measurements (Fig. 2, *A* and 4, *A*). In 18 patients, the average value of the vital capacity before the operation was 2,363 c.c. in the period free of attack. The average value after the intervention was 2,792 c.c.; hence the average rise was 18 per cent. There was an average increase in 10 cases of 20.4 per cent, no change in 2 cases, and a decrease in 6 cases. This decrease did not exceed the degree of reduction in the respiratory function that accompanies thoracotomy. Causes of the reduced vital capacity are irreversible emphysema, silicosis, deformity of the thorax, pectus carinatum, lobectomy, emphysema after postoperative spontaneous pneumothorax.

Before operation, the average pulmonary ventilation rate maximum of 8 patients was 43 L. per minute. Postoperatively the average value rose to 50 L. per minute, which meant a rise of 16 per cent. There was an increase in 2 cases, no change in one case, and a reduction in 2 cases. In the latter 2 cases, lobectomy was performed.

Concerning the question of damage, we can point out that after a systematic follow-up of the patients' condition we have not been able to observe any sign in the postoperative period that indicated any reduction of the defensive ability of the respiratory function and the lung.

*Summary of examinations.* Our examinations on the respiratory system show that the interruption of the extrinsic vegetative innervation of the asthmatic lung can be brought into relation with the cessation or the improvement of the allergic and infectious processes of the lung, on the one hand, and the spastic condition of the bronchial system, on the other hand. Parallel with the relaxation in the asthmatic bronchial spasm, the secondary changes—but, first of all, the morphologic signs of reversible emphysema—have improved, in some cases, almost entirely regressed. Changes which already have become irreversible, such as substantial emphysema and rigid thorax, have not changed essentially. According to bronchograms taken during coughing, the function of expectoration of the bronchial system has not exhibited any damage, on the contrary, it has improved considerably.

the bronchial spasm and the improve- emphysema were followed by a favor- which was expressed in higher vital maximum.

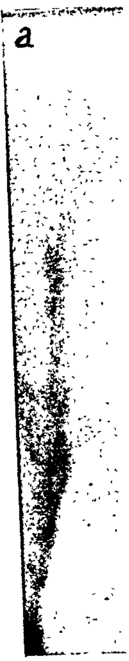


Fig.
changes t
the level
diaphragı
diaphragı

the x-rays of the t
t after left-side
blood vessels

Fig. 6.—A comparison of the bronchograms upper lol. denervation; *a*, spastic condition in the whole bronchial side It of the pressure of the opaque medium, the peripheral pulmonar nervation the spastic signs disappeared and the periphera certain extent, alveolar filling can also be observed.

aphic
:opera

physical, radiographic, and electrocardiogram examinations have justified our suppositions that the denervation of the lung in asthmatic patients has not exerted any harmful effect on the circulatory system and on the circulation itself, but it might bring improvement in the circulatory disturbances associated with an asthmatic condition (Figs. 7 and 8).

The clinical symptoms induced by the venous stasis and the change of configuration of the heart have regressed.

The electrical axis deviation owing to hypertension of the pulmonary blood circulation and the signs in the electrocardiogram of the relative myocardial ischemia, following strain of the circulation, have shown regression. We have not seen any substantial change in the pulse and the blood pressure after denervation.

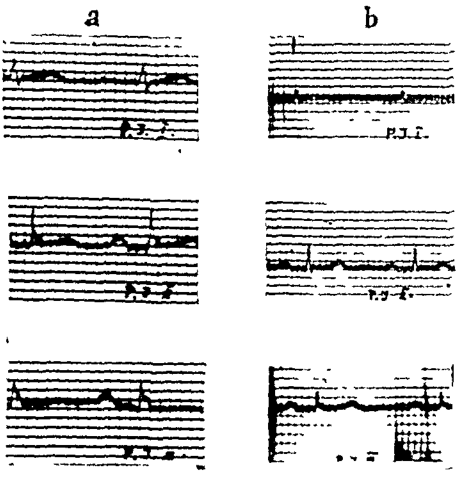


Fig. 8.—Electrocardiogram curves of P. J. *a*, Before the operation, 96-frequency sino-rhythm. $P_2$ and $P_3$ are higher and wider ($P_2$ 0.10 sec., $P_3$ 0.10 sec.). T wave is depressed in Leads I and II and it is in the isoelectric line in Lead III. *b*, After the operation, 60-frequency sino-rhythm. $P_2$ and $P_3$ became lower, their width did not change. The T wave became higher in Lead II, and in Lead III a positive T wave can be seen.

Comparison: After the operation the T waves became normal and the somewhat wider $P_2$ and $P_3$ waves became lower.

Our later observations with clinical, radiographic, and electrocardiographic examinations did not show any signs of increased use of the right side of the heart, pulmonary hypertension, or stasis of the pulmonary blood circulation.

Owing to the part played by the nervous system in bronchial asthma and to the neurosurgical character of the intervention, we have noted the neuropsychiatric status of every patient before and after operation.

A significant decrease of eosinophilia must be mentioned (Fig. 9). The results of the laboratory examinations (blood count, basal metabolism rate liver function test, serum electrolyte, among others) after the operation also support our conclusion that the denervation is not followed by any damage to the general condition of the organs.

*The Changes Produced in the Condition of the Patients.*—The most important result of the operation was that the heavy and frequent attacks accompanied by the asthmatic condition have been stopped or considerably reduced depending on the extension of denervation (uni- or bilateral), and the degree of reversibility of the secondary changes.

Considering the subjective data, the ability to work, and the findings of the objective examinations just described, we have divided the patients into three groups based on the result of the treatment.

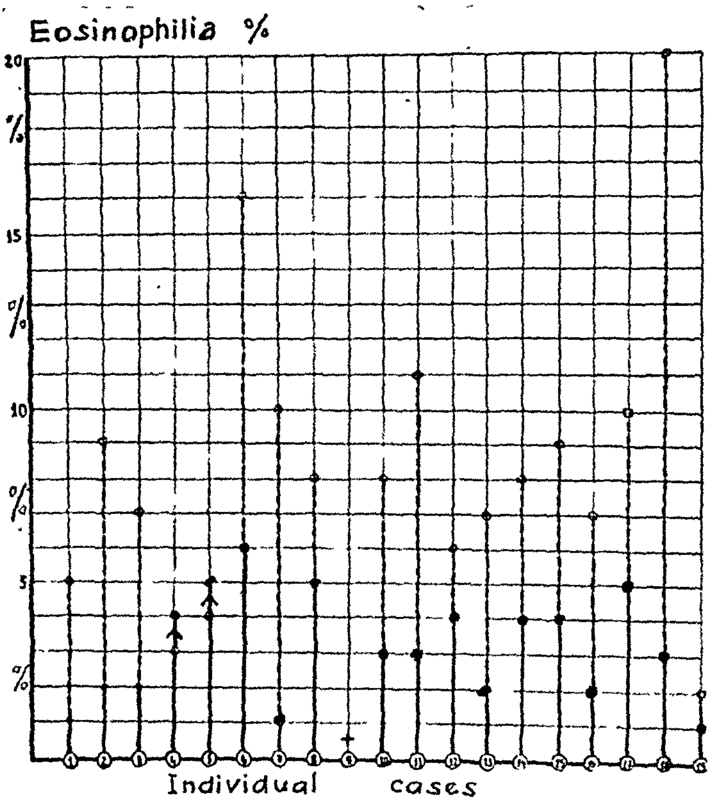


Fig. 9.—The development of the blood eosinophilia in our denervated patients. The values before the denervation are marked with a circle, after the denervation with a dot.

1. In the first group were those patients whose frequent attacks accompanied by the asthmatic condition have ceased to recur. They can continue their occupation and practically do not need any further treatment. Of 19 patients, 10 belonged to the first category. With the exception of 2 patients, they all had bilateral denervation. In this group besides the denervation, 2 patients also had lobectomy performed because of bronchiectasis.

2. In the second group were those patients in whom dyspnea or less severe asthmatic attacks, which could be easily controlled by drugs, sometimes

occurred In these cases we could not recognize an asthmatic condition postoperatively These patients were also able to return to employment but only in an easier sphere of work Seven patients belonged in this category In 2 of them bilateral and, in the other 5, unilateral denervation was performed It should be noted that in the 2 bilaterally denervated patients irreversible changes have existed previously Because of the bisystemic and bilateral innervation of the lungs, final results cannot be determined after unilateral interventions Therefore evaluation is only possible after the second operation

3 In the third category, we listed our only patient who did not improve In this case, a recurrent nerve paresis (right side) occurred as a surgical complication Although bilateral denervation was performed, improvement could not be observed

The time of follow up extends from the first intervention to one to three years That involves six seasons of the year in which the asthmatic condition usually becomes worse The time of follow up points out the lasting efficacy of the operation

Concerning the selection of time for surgical intervention, our opinion is that it is advisable to perform the operation before the secondary changes become irreversible, because in this manner, besides the disappearance of the heavy and frequent attacks accompanying the asthmatic condition, the secondary changes also regress

*Explanation of the Mechanism of Action of Denervation and Conclusions*—The therapeutic effect of the intervention can be explained with the bilateral elimination of the extrinsic vagus and sympathetic pathways of the lung Our surgical procedure interrupts more radically and therefore more efficiently the direct neural afferent and efferent connection between the vegetative centers and the lung Thus, the vicious circle of the reflex actions, playing an important part in the production of attacks, can be abolished or greatly reduced in its intensity

We have carried out examinations of the respiratory function after denervation in order to clarify the effect of the intervention

Before operation, the humoral stimulus caused by histamine has been able to provoke attacks After denervation, under the effect of histamine, we have not observed clinical signs of asthma (Figs 2 *C* and 4, *C*) Even the reduction in the values of the increased respiratory function characteristic of the attack has not occurred, the vital capacity has been reduced only 2 per cent and the pulmonary ventilation rate maximum in 9 per cent

We have been able to register the surprising continuous increase of the already well-improved respiratory function under the effect of adrenalin (Figs 2, *B* and 4, *B*) The vital capacity and the pulmonary ventilation rate maximum have shown a further 18 per cent and 22 per cent rise, respectively The question arises as to how to interpret the favorable change in the allergic and infectious processes of the lung and in the asthmatic signs and, also, how to explain the unsuccessful use of histamine and, at the same time, the better adrenalin effect after the denervation

It is well known that during the allergic and infectious processes, histamine-like materials ("H" materials: acetylcholine, histamine) have been released. These chemical mediators, as we have already stated, play an important role in the pathomechanism of the asthmatic attack.

On the basis of our observations and the data from the literature, our opinion is that the denervated asthmatic lung* reacts as if it were "more resistant" in relation to parasympathomimetic mediators producing the allergic and infectious processes or other stimuli. These chemical materials—besides the local temporary effect—were not able to produce the vicious circle of the reflex actions needed for the asthmatic paroxysm since we had interrupted the vagosympathetic pathways conducting the pathologic reflexes between the vegetative centers and the lung. The regression of the allergic and infectious processes of the lung, the normalization of its tendency to allergy and infections, and of its pathologically increased reactivity may be due to the elimination of the reflex action. The favorable change in reactivity can be further attributed to better adrenalin effect proved by us. Co-Tui has shown, with experiments in animals, that after the denervation of the lung, the bronchioles also become sensitive to adrenalin in their own circulation, in the same manner as the blood vessels. Our studies confirm the idea of an increased adrenalin sensitivity of the denervated human lung.

Finally, the chemical mediators released during the allergic and infectious processes developed after the denervation are not able to start reflex actions oducing a paroxysmal syndrome. At the same time, by means of the ineased adrenalin sensitivity of the pulmonary tissue, the antagonism to the ysiologic quantity of adrenalin circulating in the blood is able to compensate r the local effects of these materials, which are manifest in parasympathetic gns (bronchial spasm, hypersecretion, and so forth).

In physiologic conditions, the alternations of the antagonist parasympaetic and sympathetic effects are in equilibrium. The wide scale of stimuli the asthmatic lung can upset the equilibrium and produce a pathophysiogically increased parasympathetic effect responsible for the asthmatic contion. After the most radical elimination of the extrinsic innervation of the ng, the state of equilibrium becomes stabilized.

Furthermore, we came to the conclusion that in the asthmatic status, the nduction of the direct vagosympathetic pathway connecting the vegetative nter with the lung means a harmful rather than beneficial function for the ganism. Since it conducts the reflex actions leading to attack, its function changes from physiologic to pathologic. Thus, the well-known secondary changes (emphysema, strain of the circulation, among others) become worse and irreversible. In this case, we consider the elimination of the extrinsic pathways to be justified. Then it is even more beneficial because the function of the lung and the adaptability of the respiration are not exposed to damage. Our suppositions have been greatly supported by the practical observation that the biologic capacity and adaptability of our asthmatic patients have not declined, on the contrary, they have improved considerably during many years of observation.

---

*We are applying the expression of lung denervation for denoting the character of the intervention. Anatomically and physiologically the organs could not be entirely denervated.

## SUMMARY

In the pathomechanism of the asthmatic attack, we attach great importance to the reflex actions maintaining a vicious circle, transmitted between the vegetative centers and the lung by the lung's extrinsic vegetative pathways.

In cases of intractable bronchial asthma, because of the bilateral and bisystematic innervation of the lung, we have used a surgical intervention in which the afferent and efferent innervation of the lungs on both sides has been very radically interrupted (the pulmonary branches of the vagus nerve and the sympathetic ganglia of the second to fifth thoracic vertebrae have been resected).

As regards the efficiency of the operation, the results obtained as compared with the test examinations before the operation are as follows.

Of the 19 patients operated on, the results were very good in 10, and good in 7. One patient has remained unchanged. Soon after the second intervention one patient died.

Objective examinations have demonstrated that after the intervention the allergic and infectious processes of the lung have regressed, the spastic condition of the bronchi has relaxed and, along with these, the reversible secondary asthmatic changes have improved. We have not observed any damage.

During the time of observation, up to three and one half years, no regression of the good results has been observed.

The favorable effect of the operation is attributed to the stabilization of the vagosympathetic equilibrium in the asthmatic lung isolated from the vicious circle of pathogenic reflex actions. We demonstrated the resistance of the lung to "H" materials, on the one hand, and increased adrenalin sensitivity, on the other. Parallel with these results a favorable change occurs in the reactivity of the lung, that is, it becomes normal.

## REFERENCES

Abbott, O. A., Hopkins, W. A., and Guilfoil, P. H.: J. Thoracic Surg. 20: 571, 1950.
Blades, B., Beattie, L. J., Jr., and Elias, W. S.: J. Thoracic Surg. 20: 584, 1950.
Bogendorfer: Arch. f. exper. Path. u. Pharmakol. 124: 65, 1927.
Bogendorfer: Arch. f. exper. Path. u. Pharmakol. 126: 378, 1927.
Bogendorfer: Arch. f. exper. Path. u. Pharmakol. 133: 107, 1928.
Braeucker, W.: Arch. klin. Chir. 139: 1926/a.
Braeucker, W.: Arch. klin. Chir. 142: 1926/b.
Braeucker, W.: Beitr. klin. Tuberk. 66: 1, 1927.
Braeucker, W.: Nervenarzt 6: 449, 1933.
Buchwald: Med. Klin. 36: 1302, 1940.
Bulatov: Terap. Archiv 6: 25, 1953, Moscow.
Carr, D., and Chandler, H.: J. Thoracic Surg. 17: 1, 1948.
Co Tui, Burstein, C. L., and Wright, A. H.: J. Pharmacol. & Exper. Therap. 58: 33, 1936.
Danielopolu, D.: Le systeme nerveux de la vie vegetative, Paris, 1943, Doin & Cie.
Danielopolu, D.: Phylaxie paraphylaxie et maladie specifique, Paris, 1946, Masson & Cie.
De Burgh Daly, I., and von Euler, V.: Proc. Roy. Soc. London 110: 92, 1932.
De Burgh Daly, I.: A tudo mukodeset befolyasoló nehany tényezorol, Orvosi hetil. 82: 515, 1938.

Dijkstra: Beitr. z. klin. d. Tuberc. 92-1939.
Dimitrov: J. internat. chir. 13: 588, 1953.
Dzsinich, A.: Klin. Wchnschr. 15-1935.
Euler: Physiologie des Lungenkreislaufes, Dresden, 1951, Theodor Steinkopf.
Filipp, Szentiványi, and Mess. Orvosi hetil. 16-1952.
Fontaine, R., and Herrmann, L.: Arch. Surg. 16: 1153, 1928.
Friedberger-Oshikawa: Z. f. Immun. fschg. u. exper. Therap. 33: 306, 1922.
Hajós, K.-Rajka: Asthma, Ekzema, Budapest, 1944, Eggenberger.
Hebb, C. B.: J. Physiol. 99: 57, 1940.
Hetényi: Részletes belgyógyászat, Tankönyv Kiadó, 1951.
Kappis, M.: Med. Klinik. 20: 1347, 1924.
Keztyüs, et al: Elöadás az 1954, évi Pavlovi Konferencián.
Kiss: Idegrendszeri kutatásaim eredményei, Magyar Belorv. Arch. 2-1951.
Kiszelev, and Migina: Klin. med. 3-1949.
Klassen, K. P., Morton, D. R., and Curtis, G. H.: J. THORACIC SURG. 20: 552, 1950.
Kümmel: Klin. Wchnschr. 11-1923.
Kümmel: Zentralb. chir. 53-1926.
Kuntz: The Autonomic Nervous System, ed. 4, Philadelphia, 1953, Lea & Febiger.
Leriche, R., and Fontaine, R.: Presse méd. 47: 241, 1939.
Levin, G. L. L.: Ann. Surg. 102: 161, 1935.
Mansfeld: Orvosi hetil. 4: 321, 1948.
Miscall, L., and Rovenstine, E. A.: Surgery 13: 459, 1942.
Mitchell: Anatomy of the Autonomic Nervous System, Edinburgh-London, 1953, E. & S. Livingstone.
Phillips, E. W., and Scott, W. J. M.: Arch. Surg. 19: 1425, 1929.
Reitler: Wien. klin. Wchnschr. 38: 267, 1924.
Rienhoff, W. F., Jr., and Gay L. N.: Arch. Surg. 37: 456, 1938.
Szperanszkij: A Basis for the Theory of Medicine, New York, 1935, Internat., publisher.
Tsuji: Wesen und Behandlung des Bronchialasthmas, Berlin-Wien, 1939, Urban & Schwarzenberg.
Tusch, E., and Moser, H.: Ztschr. Immunätsforsch. 108: 89, 1950.
Visnyevszkij: cit. Bulatov.
White-Smithwick-Simeon: The Autonomic Nervous System; Anatomy, Physiology and Surgical Application, ed. 3, New York, 1952, The Macmillan Company.
ropajev: cit. Urbach, E., and Gottlieb: Allergy, New York, 1939.

# PULMONARY CYSTIC DISEASE PHYSIOLOGIC STUDIES AND RESULTS OF RESECTION

Arthur A. Siebens, M.D.,* Austin R. Grant, M.D.,** Donald C. Kent, Lieutenant Commander (MC) USN,*** Robert Klopstock, M.D.,**** and John J. Cincotti, M.D.*****
With the Technical Assistance of Betty L. Vestal
St. Albans and Brooklyn, N.Y.

## INTRODUCTION

IT IS among the purposes of this paper to show that resection of large pulmonary cysts in selected patients is followed by striking improvement in respiration. This favorable effect may be particularly evident in patients for whom hypoxia is one of the manifestations of impaired lung function. These results of surgery stand in contrast to those previously reported[1] in patients for whom hypoxia was interpreted to be among the contraindications to resection.

There have been relatively few published studies of the functional consequences of treating pulmonary air cysts by resection. Three articles dealing primarily with the physiology of cystic disease have appeared in the American literature.[1-3] Of the patients included in these, 5 were treated surgically, 2 by pneumonectomy, and 3 by subtotal resection.[1, 2] The scarcity of the data which came to our attention prompted a review of the pulmonary function studies of 11 men with unilateral and bilateral cystic disease. Six patients, of whom 3 were studied before and after resectional therapy, were selected as representative of the group.

Some patterns of physiologic abnormality into which these patients were placed are among those described by Baldwin and her colleagues[1]: (1) those with free bronchial communication and normal lungs, and (2) those with poor or intermittent bronchial communication and normal lungs. In addition to these recognized functional types, we have included patients in whom cystic disease was associated with poor oxygenation of arterial blood and who did

---

From the Cardiopulmonary Laboratories of the United States Naval Hospital, St. Albans, N.Y., and of the Veterans Administration Hospital, Brooklyn, N.Y., and the Thoracic Surgical Service, Veterans Administration Hospital, Brooklyn, N.Y.

This project was aided by a contract between the Office of Naval Research, Department of the Navy, and the State University of New York. Reproduction of this paper in whole or in part is permitted for any purpose of the United States Government.

Received for publication Dec. 23, 1955.

*Associate Professor of Physiology, State University of New York College of Medicine at New York.

**Assistant Chief of Surgery and Physician in Charge of Cardiopulmonary Laboratory, Veterans Administration Hospital, Brooklyn, N.Y., and Clinical Assistant Professor of Surgery.

***Chief of Cardiopulmonary Laboratory, United States Naval Hospital, St. Albans, N.Y., and Clinical Associate Professor of Surgery.

****Chief of Thoracic Surgical Service, Veterans Administration Hospital, Brooklyn, N.Y., and Clinical Associate Professor of Surgery.

*****Chief of Surgical Service, Veterans Administration Hospital, Brooklyn, N.Y., and Clinical Associate Professor of Surgery.

TABLE I. PRE- AND POSTOPERATIVE VOLUMETRIC AND VENTILATORY

| CLINICAL DATA | | | | | LUNG | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | V.C. | | | | R.V. | |
| PATIENT NO. | AGE (YRS.) | B.S.A. M² | MONTHS POSTOP. | SIDE OF CYST OR OPERATION | C.C. | % OF PRED. | RT. (%) | LT. (%) | C.C. | % OF PRED. |
| 1 | 29 | 1.91 | | Lt. | 6403 | 143 | 49 | 51 | 1547 | 139 |
| 2 | 25 | 1.66 | | Lt. | 4149 | 96 | 53 | 47 | 1551 | 143 |
| 3 | 27 | 1.96 | | Rt. | 4644 | 101 | 51 | 49 | 2588 | 225 |
| 4 | 49 | 1.66 | | Rt. | 2480 | 68 | | | 1546 | 139 |
| | | | 2½ | Rt. | 3270 | 90 | | | 1205 | 108 |
| 5 | 43 | 1.84 | | Bilat. | 4026 | 99 | * | * | 2295 | 185 |
| | | | 3¾ | Lt. | 3115 | 77 | 65 | 35 | 1421 | 115 |
| | | | 3¾ | Rt. | 2738 | 67 | 43 | 57 | 1383 | 112 |
| 6 | 31 | 1.80 | | Bilat. | 1876 | 45 | 38 | 62 | 1091 | 104 |
| | | | 1⅓ | Rt. | 2466 | 59 | | | 1458 | 138 |
| | | | 2½ | Lt. | 2936 | 70 | 54 | 46 | 1323 | 126 |

*Patient unable to perform vital capacity during bronchospirometry.

not suffer from advanced pulmonary emphysema. These are to be distinguished from a group of patients identified by Baldwin[1] in whom the abnormalities of cystic disease were compounded by advanced emphysema.

Cystic disease of the lung is rivaled by few pulmonary lesions in producing a variety of respiratory abnormalities. The number of patients included in this paper was restricted in order to permit discussion of each study individually. The tabular data are supplemented by graphs permitting ready recognition of changes and departures from normal. One of the objectives of this study was, therefore, to illustrate the application and interpretation of function data.

## MATERIAL AND METHODS

Clinical data pertaining to the 6 male patients are included in Table I.

The pulmonary function was studied using conventional methods. The maximum eathing capacity, performed in the sitting position, was measured by collecting expired air a Douglas bag and passing the contents through a gas meter. The best of three fifteen-cond attempts was recorded. The subdivisions of the vital capacity were inscribed during osed-circuit spirometry. The timed vital capacity was obtained by measuring the proportion the vital capacity delivered in one, two, and three seconds of forceful expiration. The nctional residual capacity was measured by a modification of the Darling open-circuit ethod,[4] the figures recorded being the mean of two analyses which checked within 10 per cent. The residual volume was calculated by subtracting the expiratory reserve from this mean value. The alveolar index, the concentration of nitrogen in "alveolar air" after the patient has breathed 100 per cent oxygen for seven minutes, was also measured in duplicate, the mean of the two values appearing in the table.

Bronchospirometry was performed through a Carlens' catheter after topical anesthesia of pharynx, larynx, and trachea with 1 per cent Pontocaine. The total capacity of each lung individually was measured using Darling circuits adapted for simultaneous unilateral determinations.[5] All of the lung volumes were converted to body temperature, ambient pressure, and saturation with water vapor (BTPS).

Blood gas contents and the concentration of nitrogen in air samples were measured using the method of Van Slyke and Neill.[6] Blood gas tensions were measured by the method of Riley.[7, 8] The composition of expired gases was determined with the Scholander apparatus.[9]

DATA IN SIX MEN WITH CYSTIC DISEASE OF THE LUNG

| VOLUMES | | | | | INTRAPULMONARY MIXING | VENTILATION | | | | | | | OXYGEN UPTAKE | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| T.L.C. | | F.R.C. | R.V./ T.L.C. (%) | F.R.C./ T.L.C. (%) | | M.B.C. | | TIMED V.C. PER CENT | | | AT REST | | | |
| C.C. | % OF PRED. | C.C. | | | VOLS. % $N_2$ | L./ MIN. | % OF PRED. | 1 SEC. | 2 SEC. | 3 SEC. | RT. (%) | LT. (%) | LT. (%) | LT. (%) |
| 7950 | 142 | 3932 | 19 | 49 | 6.29 | 159 | 117 | 86 | 100 | 100 | 48 | 52 | 54 | 46 |
| 5700 | 105 | 2718 | 27 | 48 | 4.08 | 121 | 99 | 68 | 80 | 85 | 62 | 38 | 65 | 35 |
| 7232 | 126 | 3738 | 36 | 52 | 4.94 | 128 | 90 | 54 | 65 | 73 | 48 | 52 | 8 | 92 |
| 4026 | 85 | 1997 | 38 | 50 | 1.43 | 56 | 55 | 50 | 65 | 70 | | | | |
| 4475 | 94 | 1906 | 27 | 43 | 0.69 | 67 | 66 | 66 | 78 | 88 | | | | |
| 6321 | 119 | 3042 | 36 | 48 | 0.75 | 109 | 92 | 62 | 78 | 85 | 56 | 44 | 65 | 35 |
| 4536 | 86 | 2062 | 31 | 45 | 2.47 | 82 | 69 | 58 | 76 | 79 | 60 | 40 | 67 | 33 |
| 4121 | 78 | 1982 | 34 | 48 | 0.69 | 132 | 112 | 84 | 92 | 100 | 47 | 53 | 43 | 57 |
| 2967 | 56 | 1091 | 37 | 37 | 2.76 | 80 | 63 | 79 | 90 | 94 | 39 | 61 | 38 | 62 |
| 3924 | 75 | 2035 | 37 | 52 | 2.75 | 136 | 107 | 85 | 89 | 93 | | | | |
| 4259 | 81 | 1950 | 31 | 46 | 0.72 | 108 | 85 | | | | 60 | 40 | 57 | 43 |

The methods of Lilienthal and co-workers,[10] and Riley and his colleagues[11, 12] were used in measuring the pulmonary diffusing capacity[14] and the ventilation perfusion relationships. The latter term refers to the *distribution* of blood and gas within the lungs and is expressed in terms of physiologic dead space ($V_D/V_T$) and venous admixture ($\dot{Q}_{va}/\dot{Q}_t$). A high dead space (exceeding 30 per cent of the tidal volume) indicates that an unusually large proportion of air entering the respiratory tract is distributed to nonperfused or poorly perfused areas. A patient with occlusion of a branch of the pulmonary artery would have an elevated dead space. The measurement is based on the assumption that expired air can be considered to be made up of two fractions, one having the carbon dioxide content of inspired air and originating in dead space and the other having the carbon dioxide content of alveolar gas. The dead space ratio indicates the proportions of these two gases which, if mixed, would result in gas with the composition of expired air.

The venous admixture ratio is similar in concept. One assumes that peripheral arterial blood is a mixture of two streams, one being mixed venous blood and the other blood in equilibrium with alveolar air. In normal individuals, the saturation of arterial blood is equal to that of a mixture containing 6 parts or less of mixed venous blood and 94 parts or more of blood in equilibrium with alveolar gas. Venous admixture ratios in excess of 6 per cent indicate that an unusually large proportion of the right cardiac output is distributed to unventilated or poorly ventilated areas. In calculating the venous admixture ratios of the present patients, mixed venous blood was assumed to be 25 per cent less saturated than pulmonary capillary blood when the patient was at rest and 40 per cent less saturated during treadmill exercise.

Oxygen diffuses from alveolar gas into pulmonary capillary blood because the pressure of oxygen in the alveolus ($P_{A_{O_2}}$) exceeds that in the pulmonary capillary ($P_{C_{O_2}}$). The diffusing capacity of the lung is the volume of oxygen ($\dot{V}_{O_2}$) which diffuses across the alveolar capillary membrane every minute per unit of pressure difference between the two sides of this membrane. It is calculated by dividing the oxygen uptake ($\dot{V}_{O_2}$) by a mean value for the pressure difference in mm. Hg between the two sides of this membrane ($\overline{P_{A_{O_2}}-P_{C_{O_2}}}$). It is low either when the dimensions of the alveolar capillary bed have been reduced, as after pulmonary resection,[14] or/and when the membrane becomes less permeable to oxygen, as in the granulomatous diseases.[15] In the absence of the latter, it is an index of the size of the alveolar capillary bed and is interpreted as such in this paper.

When one exercises, the diffusing capacity increases, reaching a maximum value when the oxygen uptake approaches 1,200 c.c. per minute. Under these circumstances the dimensions of the alveolar capillary bed approximate their maximum value. The maximum diffusing capacities of 9 young men with minimal or no demonstrable pulmonary pathology studied at the St. Albans laboratory[14] ranged from 68 to 86 with a mean of 76.

TABLE II. PRE- AND POSTOPERATIVE GAS EXCHANGE

| | | | ANALYTICAL | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| PATIENT NO. | PRE- OR POSTOP. | ACTIVITY | $S_{aO_2}$ (%) | $P_{aO_2}$ (MM. Hg) | $C_{aCO_2}$ (VOLS. %) | $P_{aCO_2}$ (MM. Hg) | BP-47 (MM. Hg) | $\dot{V}$ (L./MIN.) | $V_T$ (C.C.) |
| 1 | Pre | Rest | 96.1 | 91 | 51.41 | 41 | 722 | 8.9 | 533 |
| | | Exercise | 96.3 | 94 | 43.40 | 42 | 722 | 40.8 | 1571 |
| | | Exercise | 78.3 | 43 | 37.87 | 37 | 722 | 67.2 | |
| 2 | Pre | Rest | 97.2 | 91 | 51.06 | 41 | 714 | 6.7 | 404 |
| 3 | Pre | Rest | 94.8 | 83 | 38.77 | 35 | 705 | 11.0 | 449 |
| 4 | Pre | Rest | 90.9 | 70 | 48.45 | 45 | 709 | 9.2 | 551 |
| | | Exercise | 89.2 | | | | | | |
| | Post | Rest | 94.9 | 83 | 50.01 | 43 | 722 | 14.2 | 835 |
| | | Exercise | 91.1 | | | | | | |
| 5 | Pre | Rest | 94.8 | 86 | 42.96 | 35 | 712 | 8.1 | 1071 |
| | | Exercise | 96.1 | | | | | | |
| | Post I | Rest | 93.5 | 78 | 43.94 | 37 | 707 | 6.9 | 691 |
| | | Exercise | 93.9 | | | | | | |
| | Post II | Rest | 96.6 | 93 | 45.72 | 44 | 723 | 8.4 | 704 |
| | | Exercise | 93.9 | | | | | | |
| 6 | Pre | Rest | 92.0 | 69 | 46.92 | 35 | 717 | 7.9 | 265 |
| | | Exercise | 88.0 | 58 | 39.99 | 28 | 717 | 44.0 | 1190 |
| | | Exercise | 67.0 | 34 | 36.65 | 29 | 717 | 47.8 | 1290 |
| | Post I | Rest | 95.3 | 83 | 50.20 | 44 | 711 | 7.1 | 420 |
| | | Exercise | 94.9 | 83 | 44.47 | 39 | 711 | 42.1 | 1212 |
| | | Exercise | 70.3 | 40 | 38.17 | 36 | 711 | 49.3 | 1333 |
| | Post II | Rest | 95.3 | 86 | 51.18 | 46 | 703 | 6.3 | 365 |
| | | Exercise | 94.9 | 86 | 42.82 | 40 | 703 | 40.0 | 1160 |
| | | Exercise | 66.6 | 39 | 34.49 | 36 | 703 | 55.6 | 1333 |

The following conventional abbreviations[16] appear in Tables I and II:

B.S.A., $M^2$—Body surface area in square meters
V.C.—Vital capacity
R.V.—Residual volume
T.L.C.—Total lung capacity
F.R.C.—Functional residual capacity
R.V./T.L.C.—Ratio of the residual volume to the total lung capacity in per cent
F.R.C./T.L.C.—Ratio of the functional residual capacity to the total lung capacity in per cent
M.B.C., L./min.—Maximum breathing capacity in liters per minute, BTPS
$S_{aO_2}$, %—Oxyhemoglobin saturation of arterial blood in per cent
$P_{aO_2}$, mm. Hg—Partial pressure of oxygen in arterial blood in millimeters of mercury
$C_{aCO_2}$, Vols. %—c.c. of carbon dioxide contained in 100 c.c. of arterial blood
$P_{aCO_2}$, *mm. Hg—Partial pressure of carbon dioxide in arterial blood in millimeters of* mercury
BP-47, mm. Hg—Barometric pressure minus correction for water vapor tension of air saturated at body temperature, in millimeters of mercury
$\dot{V}$, L./min.—Total ventilation in liters per minute, BTPS
$V_T$, c.c.—Tidal volume in c.c., BTPS
$F_{eCO_2}$, %—Concentration of carbon dioxide in expired air in per cent
R—The gas exchange ratio, or respiratory quotient, i.e., carbon dioxide output divided by the oxygen consumption

DATA IN SIX MEN WITH CYSTIC DISEASE OF THE LUNGS

| DATA | | | | DERIVED VALUES | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | DISTRIBUTION | | DIFFUSION | |
| | | | | | | | | MAX. DIFF. CAP. | |
| $F_{E_{CO_2}}$ (%) | $F_{I_{O_2}}$ (%) | R | $\dot{V}_{O_2}$ (C.C./MIN.) | $P_{A_{O_2}}$-$P_{a_{O_2}}$ (MM. Hg) | $\overline{P_{A_{O_2}}\text{-}P_{c_{O_2}}}$ (MM. Hg) | $V'_D/V_T$ (%) | $\dot{Q}_{va}/\dot{Q}_t$ (%) | TOTAL | M² |
| 3.241 | 20.93 | 0.76 | 315 | 9 | | 30 | 4 | | |
| 4.255 | 20.93 | 0.94 | 1546 | 12 | 31 | 22 | 3 | | |
| 3.359 | 13.15 | 1.12 | 1685 | 19 | 31 | | 3 | 54 | 28 |
| 3.523 | 20.93 | 0.84 | 234 | 11 | | 21 | 7 | | |
| 2.762 | 20.93 | 0.75 | 331 | 22 | | 31 | 10 | | |
| 3.284 | 20.93<br>20.93 | 0.85 | 292 | 26 | | 36 | 19 | | |
| 3.337 | 20.93<br>20.93 | 0.84 | 471 | 19 | | 36 | 10 | | |
| 3.292 | 20.93<br>20.93 | 0.80 | 276 | 21 | | 26 | 9 | | |
| 3.352 | 20.93<br>20.93 | 0.77 | 249 | 25 | | 26 | 15 | | |
| 2.933 | 20.93<br>20.93 | 0.84 | 247 | 8 | | 42 | 3 | | |
| 2.591 | 20.93 | 0.87 | 197 | 42 | | 29 | 24 | | |
| 3.668 | 20.93 | 0.98 | 1362 | 63 | 45 | 56 | 26 | | |
| 2.973 | 14.26 | 1.08 | 1065 | 41 | 45 | 32 | 26 | 24 | 13 |
| 2.852 | 20.93 | 0.87 | 193 | 16 | | 22 | 8 | | |
| 3.600 | 20.93 | 0.91 | 1376 | 23 | 30 | 29 | 7 | | |
| 3.141 | 13.44 | 1.07 | 1201 | 22 | 30 | 27 | 7 | 40 | 22 |
| 3.188 | 20.93 | 0.76 | 214 | 3 | | 34 | 2 | | |
| 3.770 | 20.93 | 0.85 | 1442 | 16 | 31 | 29 | 6 | | |
| 3.164 | 13.05 | 1.17 | 1224 | 22 | 31 | 33 | 6 | 40 | 22 |

$\dot{V}_{O_2}$, c.c./min.—Oxygen uptake in c.c. per minute, STPD

$P_{A_{O_2}}$-$P_{a_{O_2}}$, mm. Hg—Oxygen partial pressure difference in millimeters of mercury between "alveolar air" and peripheral arterial blood

$\overline{P_{A_{O_2}}\text{-}P_{c_{O_2}}}$, mm. Hg—Mean oxygen pressure difference between alveolar air and pulmonary capillary blood

$V'_D/V_T$, %—Ratio of dead space to tidal volume in per cent

$\dot{Q}_{va}/\dot{Q}_t$, %—Ratio of the venous admixture to cardiac output in per cent

Max. Diff. Cap.—The maximum diffusing capacity

### PATIENT NUMBER ONE

*Clinical Summary.*—This 29-year-old white man was admitted for study after pulmonary pathology had been disclosed by routine chest roentgenograms. An infection with histoplasmosis had been established by gastric smear two years previously. He was asymptomatic without cough, dyspnea, chest pain, or hemoptysis.

Physical examination revealed normal resonance and breath sounds throughout the chest. A roentgenogram (Fig. 1*A*) showed infiltrative lesions in the apical and posterior segments of the upper lobe of the right lung. Cystic changes were apparent in the left lung with d [illegible]ment

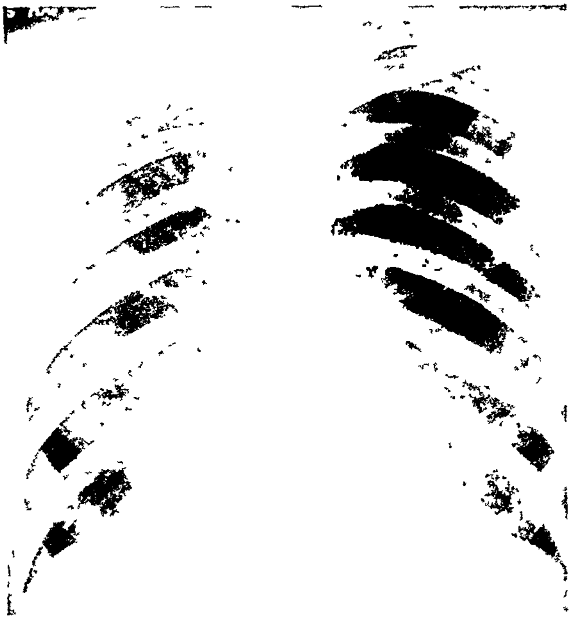


Fig. 1A.—Chest roentgenogram. Note infiltrative lesion of right upper lung field and increased radiolucency of left upper hemithorax.

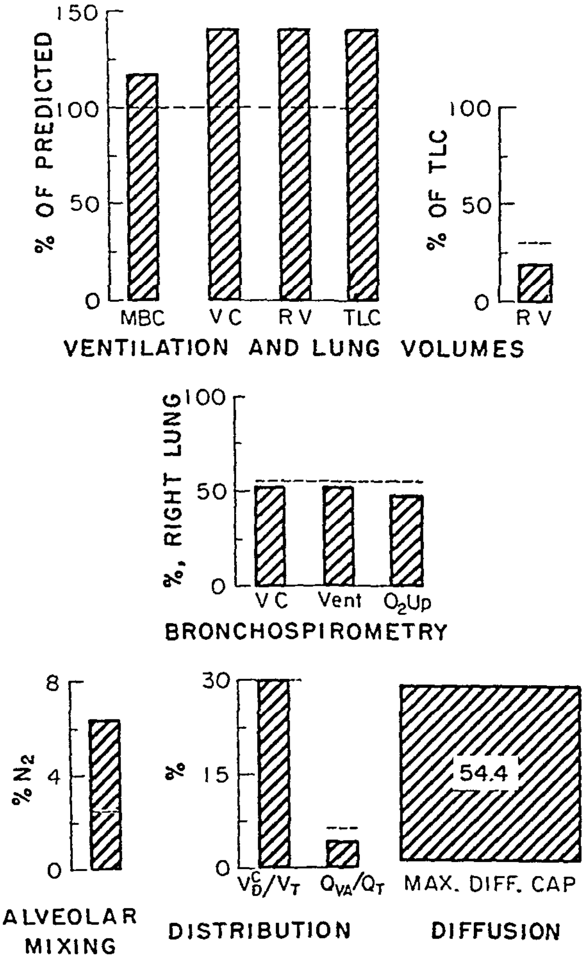


Fig. 1B—Function data of Patient 1. Note absence of large abnormalities except in index of alveolar mixing.

of the hilar markings. Tomograms demonstrated multiple air containing cysts in the left upper lobe area and compression of the left lower lobe. Pulmonary function studies were obtained (Fig. 1*B*).

During hospitalization he remained well. Since it was the opinion of the medical staff that surgery was not indicated, he was discharged.

*Physiologic Studies.*—As shown in Tables I and II and in Fig. 1*B*, measurements of the maximum breathing capacity and the lung volumes indicated no abnormality. The studies of unilateral function also failed to reveal noteworthy deficit, for the contribution of the left lung to the vital capacity, ventilation, and oxygen uptake was normal. This normal participation indicated that interference by the cyst and parenchymal infiltrate affected the left and right lungs proportionately. By reducing the performance of the right lung the infiltrative lesion could well have resulted in increasing to normal limits the relative contribution of the left lung despite some interference with unilateral function attributable to the cyst.

Ventilation perfusion relationships were also normal as indicated by the dead space ($V^{e}_{D}/V_{T}$) and venous admixture ($Q_{va}/Q_{t}$) ratios. The maximum diffusing capacity was minimally reduced—a finding consistent with decreased permeability of the alveolar capillary membrane to oxygen and/or with reduced dimensions of the capillary bed in the limited area of infiltration on the right. The nearly normal figure for this measurement, therefore, supports the ventilatory, volumetric, and distribution data in indicating no gross physiologic change attributable to the presence of the cyst.

The index of alveolar mixing was strikingly abnormal (6.296 per cent). In the absence of an elevated residual volume or of an obstructive ventilatory deficit, the most likely explanation for this finding was the presence of a comparatively patent communication between the cyst and the tracheobronchial tree. The incomplete elimination of nitrogen from the cyst during the seven minutes for which the patient breathed virtually pure oxygen was explained by assuming that the tidal exchanges of air between the cyst and the bronchial tree during quiet breathing were small. This explanation was supported by the normal dead space ratio, for this ratio would have been abnormally high had a sizeable structure not participating in gas exchange been well ventilated.

*Summary.*—This patient was selected for demonstrating that a large air cyst may interfere minimally with tests of respiration. Ventilation, lung volumes, bronchospirometry, ventilation perfusion relationships, and the maximum diffusing capacity were normal or nearly normal. Only the index of alveolar mixing was patently abnormal.

## PATIENT NUMBER TWO

*Clinical Summary.*—This 26 year old Negro man was admitted for evaluation of his pulmonary status. Three years previously a routine chest roentgenogram had revealed the presence of a cyst of the left lung in which no change had been detected by yearly studies. He denied cough, dyspnea, orthopnea, hemoptysis, or chest pain.

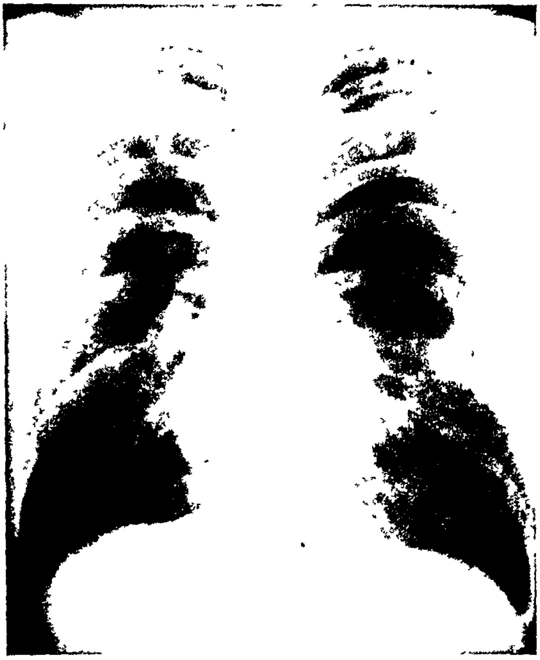


*Fig. 2A.*—Chest roentgenogram. Note oval radiolucent area in left upper lung field.

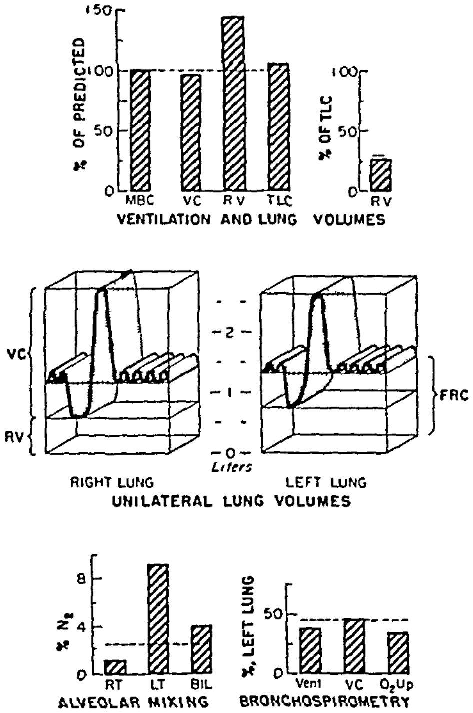


Fig. 2*B*.—Preoperative function data of Patient 2. The impairment of the left lung is shown by the slope of the expiratory tracing, the elevated residual volume and functional residual capacity, the poor alveolar mixing of respiratory gases, and the diminished contribution to ventilation and oxygen uptake. Maximum breathing capacity and bilateral lung volumes are normal.

On physical examination there were amphoric whispered-voice sounds over the second left intercostal space medial to the anterior axillary line. Chest roentgenograms showed absence of the lung markings in the upper left hemithorax (Fig. 2A). The left leaf of the diaphragm was depressed. Pulmonary function studies were obtained (Fig. 2*B*).

On Sept. 14, 1954, a left thoracotomy disclosed emphysematous and cystic changes of the posterior segment of the left upper lobe. The cystic areas were so markedly distended that the remainder of the upper lobe and the superior and posterior basilar segments of the lower lobe were compressed. The cystic and emphysematous areas were removed by wedge excision. Bronchial communications to the upper lobe cyst were demonstrated.

Three months following surgery the patient was returned to full duty.

*Physiologic Studies* (Fig. 2*B*).—Although the vital capacity was moderately slowed, the maximum breathing capacity was normal. Thus, in spite of evidence for increased resistance to the flow of air during forced expiration, the maximum voluntary ventilation was within the predicted range. Ventilation on the side of the cyst was 38 per cent of the total as compared to a normal of 45 per cent.

The lung volumes were virtually normal. Comparison of the spirometric tracings of each lung (Fig. 2*B*) showed slowing of expiration on the left side during delivery of the expiratory reserve. This unilateral obstruction possibly could be attributed to interference of the bronchospirometric catheter with air flow, to expression of air from the cyst through a narrow communication, to pressure of the cyst on adjacent parenchyma and airways causing trapping in areas distal to the point of compression, or to a combination of these factors. The placement of the catheter appeared to be optimal as judged by fluoroscopy, bronchospirometric tracings and correspondence between unilateral and bilateral volumetric measurements. The high index of alveolar mixing on the left (9.175 per cent as compared to 1.270 per cent on the right) suggested a communication which was later identified by examination of the resected specimen. Parenchymal compression was demonstrated by roentgenography and confirmed at thoracotomy. It is reasonable therefore to ascribe the prolonged expiration to the effects of the cyst rather than to artifacts of recording.

Obstructive breathing commonly results in elevation of the mid position. Correspondingly the functional residual capacity and residual volume on the left were higher than on the right side, the opposite of the normal relationship.

The elevation in the bilateral index of alveolar mixing (4.076 per cent) was attributable to mixture in the trachea of gases of unequal nitrogen concentration contributed by the two main stem bronchi.

The presence of a poorly ventilated cyst complicates determining the functional residual capacity by the Darling technique, for nitrogen elimination from the lungs, which is the basis of the method, is [illegible] by ventilation. It was likely in the present patient that the pe
nitrogen, indicated by the high alveolar index, was not re
left lung as a whole but only of the cystic lobe. In order to a

introduced by using an artificially elevated value, the mixing index observed for the normal right lung was used in calculating the functional residual capacity of the left lung. This substitution gave a figure 251 c.c. (16.1 per cent) below that obtained by using the high index actually measured. Selection of a figure for the alveolar index was inevitably arbitrary, for there was in fact no index representative of the whole lung but a group of unknown, but widely divergent, indices from pulmonary areas contributing differently to ventilation. By using the index chosen, the sum of the functional residual capacities of the two lungs (2,503 c.c.) fell within 8 per cent of the mean of two bilateral measurements (2,718 c.c.) also computed using a normal index.

Finally, it should be observed that the functional residual capacity and residual volume computed according to these assumptions were minimal figures; although the proportion contributed by the parenchyma was reasonably accurately represented, the volumes of the cysts were underestimated.

Both the physiologic dead space ($V^c{}_D/V_T$) and the venous admixture ($\dot{Q}_{va}/\dot{Q}_t$) ratios were normal indicating no gross abnormalities in the relationship between alveolar ventilation and perfusion. Pertinent to this finding was the observation that the oxygen uptake on the left (35 per cent of the total) was reduced in proportion to the reduction in ventilation (38 per cent). Inasmuch as unilateral oxygen uptake is an approximate index of the distribution of blood flow between the two lungs, the proportional reductions in unilateral ventilation and oxygen uptake would be expected in the presence of normal ventilation-perfusion relationships. Two additional inferences were drawn from the fact that ventilation-perfusion relationships were normal: (1) The normal dead space suggested that the cyst (the walls of which were doubtless negligibly perfused) was poorly ventilated. This observation was in agreement with the fact, cited above, that even after the patient had breathed virtually pure oxygen for nine minutes the air contained in the cyst still had a high concentration of nitrogen. (2) The virtually normal alveolar-arterial oxygen partial pressure difference and ratio of venous admixture to total pulmonary flow suggested that the cyst was not compressing unusually large areas of adjacent pulmonary parenchyma.

*Summary.*—Bronchospirometry was particularly useful in evaluating this patient's respiratory deficit. Slowing of expiration and impaired intrapulmonary mixing of respiratory gases, both of which were demonstrated by the bilateral studies, were shown to be localized to the side of the lesion. There were, in addition, unilateral abnormalities of ventilation, lung volumes, and oxygen uptake not identified by bilateral measurements. The unilateral data were used to illustrate the difficulties of interpreting measurements of the functional residual capacity in the presence of a communicating cyst. Ventilation-perfusion relationships were normal. The preoperative prediction that the cyst was a communicating structure was confirmed at thoracotomy.

### PATIENT NUMBER THREE

*Clinical Summary.*—This 26-year-old Negro male laborer entered the hospital complaining of a nonproductive cough of one year's duration and progressive exertional dyspnea of three years' duration. There had been two recent episodes of upper respiratory tract infection with fever, chills, and productive cough without hemoptysis. Walking one block or climbing one flight of stairs produced dyspnea.

Physical examination disclosed an essentially symmetrical chest with hyperresonance and decreased breath sounds at the right apex. There were fine inspiratory and expiratory wheezes throughout both lung fields with prolongation of the time required for expiration. There was no cyanosis, clubbing, or edema of the extremities.

A roentgenogram of the chest outlined a "large area of emphysema bounded by fine linear densities in the right upper lung field extending from the apex to the level of the hilum" (Fig 3A). Pulmonary function studies were obtained (Fig 3C).

On Aug 5, 1954, a right thoracotomy exposed a large 20 by 15 cm air containing cyst occupying the apical and posterior segments of the upper lobe of the right lung. There was marked compression and partial atelectasis of the lower and middle lobes. The cyst could be easily deflated and inflated, demonstrating free bronchial communication. Because of numerous small bullae present along the margin of the anterior segment, the cyst was removed by a right upper lobectomy. The volume of the hemithorax was tailored to the volume and shape of the remaining right middle and lower lobes by creating a new roof (pleural tent)[17] for the pleural cavity from a stripped portion of the cupola.

Three months following surgery, the patient had noted marked improvement of dyspnea.

*Physiologic Studies*.—The most striking abnormalities presented in Fig 3C are a high residual volume, poor alveolar mixing of respiratory gases, and impaired ability to expire forcefully and rapidly (timed vital capacity). These could all be accounted for by assuming that the cyst communicated poorly, or that it obstructed ventilation of ipsilateral parenchyma, or that generalized emphysema existed. A postoperative study would have differentiated among these possibilities, such, however, could not be obtained. The relatively good maximum breathing capacity did not suggest widespread generalized emphysema. There were, however, indications that the cyst was ventilated and that the function of ipsilateral parenchyma was compromised. At bronchospirometry, the contribution of the right lung to ventilation was found to be better than to oxygen uptake, showing that considerable unperfused, presumably cystic, tissue was being ventilated. Correspondingly, the dead space ratio ($V^{c}_{D}/V_{T}$) was elevated, though minimally so. The poor contribution to oxygen uptake suggested severely restricted right pulmonary flow, the normal saturation of arterial blood indicating that this flow was distributed for the most part to well-ventilated alveoli. The relative hypoventilation of some perfused areas, however, was shown by the moderately elevated venous admixture ratio ($Q_{va}/Q_{t}$). Together with bronchospirometry, therefore, studies of distribution were compatible with ventilation of cysts and restriction of gas exchange in ipsilateral parenchyma.

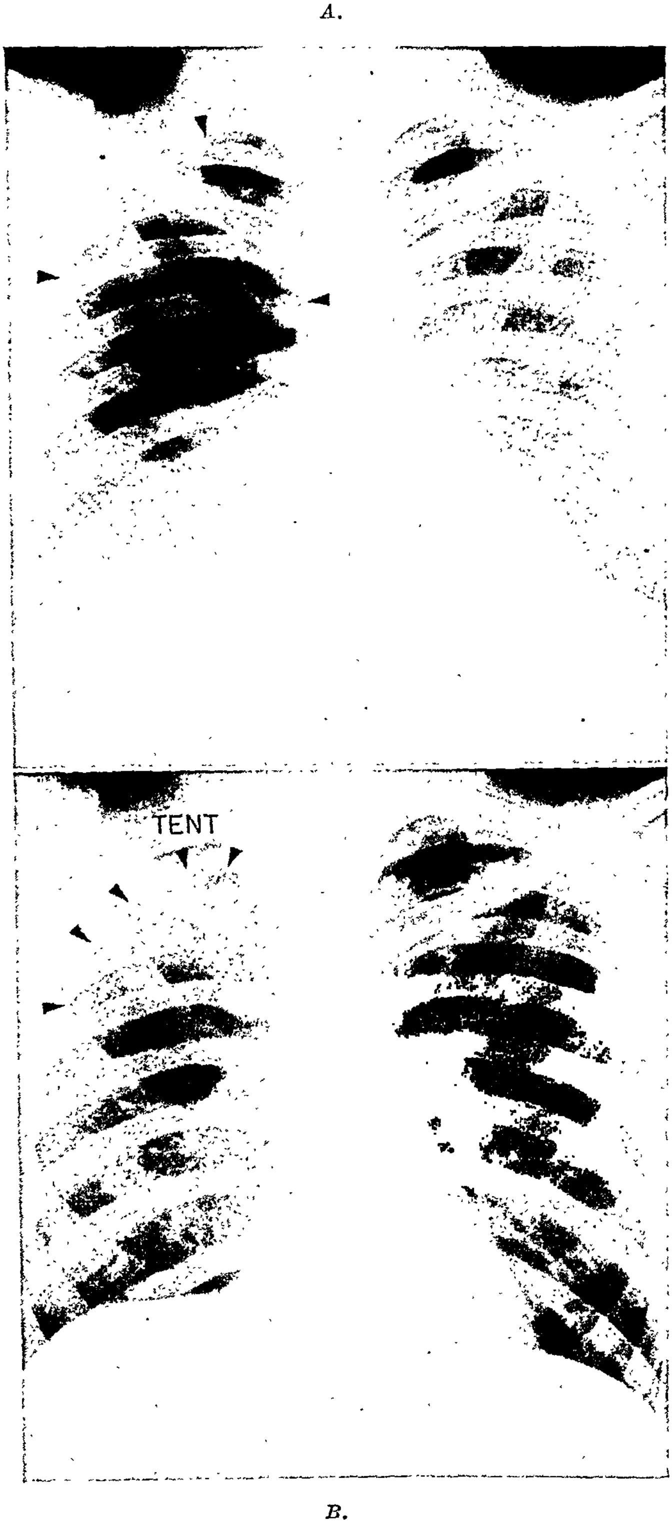


Fig. 3.—*A*, Chest roentgenogram. Note radiolucent area in right upper lung field and density in right lower lung field.

*B*, Chest roentgenogram five weeks following excision of right lung cyst and construction of pleural tent.

In addition, there was hyperventilation. Shortness of breath had been among the patient's complaints. At rest the ventilation per square meter of body surface area was 5.7 L. (normal 3.0 L.). The walking ventilation per square meter was 19 L. (normal 11 L.). As would be expected in a patient with a normal metabolic rate and an abnormally high ventilation the ventilatory equivalent was unusually high (3.4 as compared to a normal of 2.6), a characteristic of so called "inefficient" respiration. Low blood carbon dioxide tensions and contents (Table II) indicated that this hyperventilation was not restricted to unperfused tissue. In the absence of cardiac disease, hypercapnea, or hypoxia, the cause of the hyperpnea and sensation of breathlessness remained obscure.

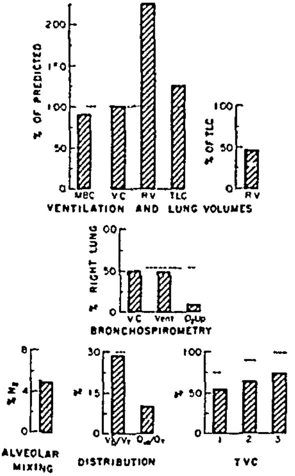


Fig. 3C.—Preoperative function data of Patient 3. The residual volume is high, mixing of respiratory gases within the lung is poor, the dead space and venous admixture ratios are both elevated, and the timed vital capacity is slowed. The contribution of the right lung to oxygen uptake is extremely low.

*Summary.*—Bilateral studies revealed a high residual volume, a slowed timed vital capacity, and poor intrapulmonary mixing of gases. There was minimal interference with ventilation on the side of the cyst but a pronounced limitation of oxygen uptake. Together with elevated dead space and venous admixture ratios, these abnormalities indicated interference with the function of the ipsilateral parenchyma. Hyperpnea at rest and exercise and the consequently low carbon dioxide tensions and contents in arterial blood remained unexplained.

PATIENT NUMBER FOUR

*Clinical Summary.*—This 49 year old white male postal clerk was admitted for progressive episodic dyspnea, palpitations, and right chest pain of three years' duration. These symptoms, occurring only on exertion, were increasing

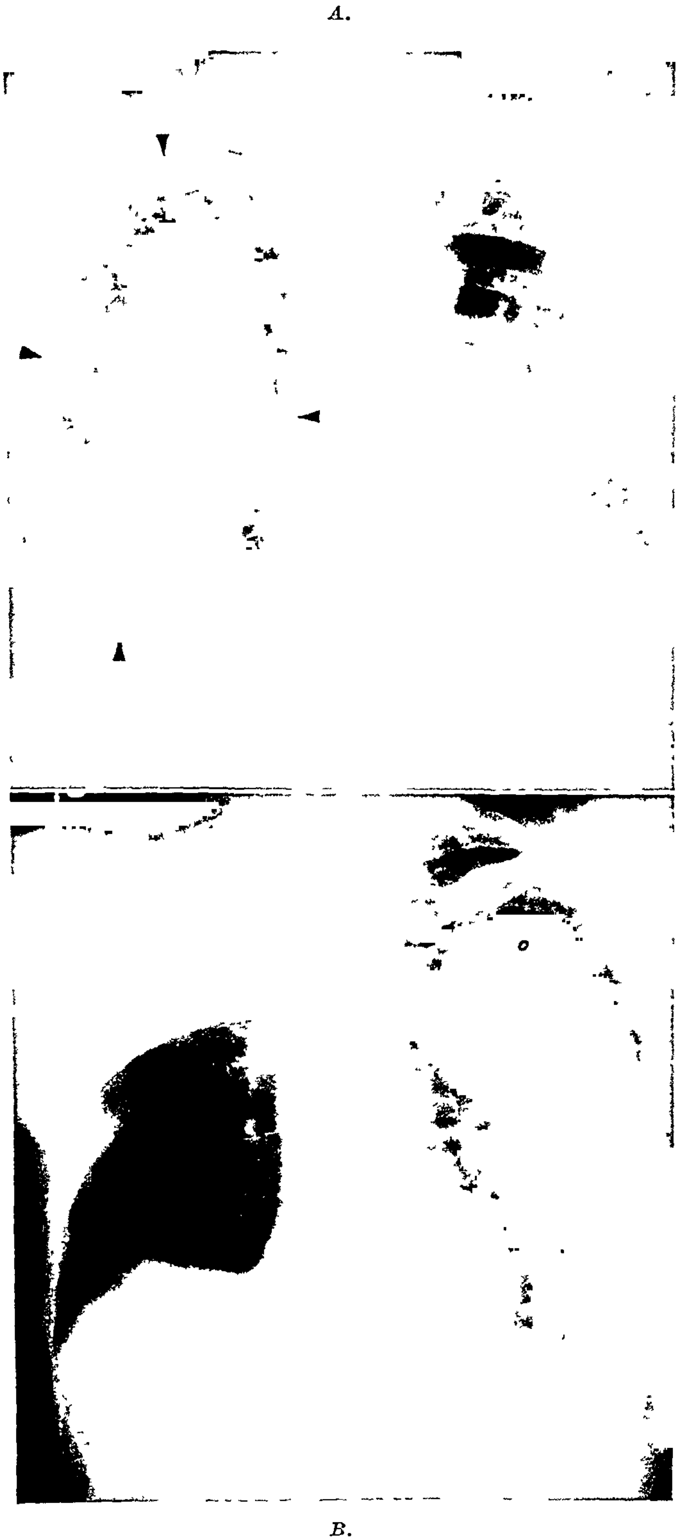


Fig. 4.—*A*, Chest roentgenogram. Note the increased radiolucency of the entire right hemithorax.

*B*, Chest roentgenogram five months following excision of right upper lobe cyst and construction of pleural tent.

in frequency from once a week originally to once a day on admission. He had had a chronic cough of 20 years' duration, productive currently of 50 c.c. of white sputum per day.

Physical examination revealed diminished respiratory excursion, increased resonance, and decreased breath sounds throughout the right chest In the left lung there was prolongation of expiration with expiratory wheezes There was no clubbing, cyanosis, or edema of the extremities

X-ray of the chest showed almost complete loss of the usual right lung markings, these being replaced centrally by dense fibrotic-like strands (Fig. 4A). The left lung showed localized increased radiolucency in the apical area The mediastinum shifted to the affected side on inspiration and to the opposite side on expiration. Pulmonary function studies were obtained (Fig. 4C)

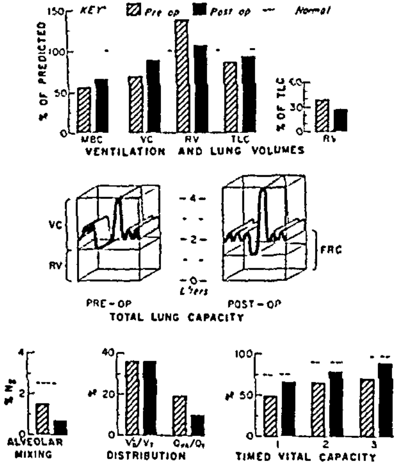


Fig 4C—Pre- and postoperative function data of Patient 4 Preoperatively there were abnormalities of ventilation lung volumes, and ventilation-perfusion relationships Severe obstruction was indicated by the low maximum breathing capacity, the slope of the expiratory tracing and the slowed timed vital capacity The normal mixing index suggested poor communication Most abnormalities were diminished by operation

On April 28, 1955, a right thoracotomy revealed a cyst originating from the upper lobe and completely collapsing the middle and lower lobes The cyst could be inflated but would deflate only with extreme difficulty. Since there were also numerous bullae of the apical and posterior segments, the cyst and the right upper lobe were resected Inasmuch as the remaining pulmonary parenchyma on expansion only partially filled the pleural cavity, the hemithorax was tailored, as described in Case No 3, with a pleural tent (Fig. 4B)

The postoperative course was uncomplicated Function studies were repeated ten weeks following resection (Fig 4C).

*Physiologic Studies.*—This patient's large cyst was associated with abnormalities of lung volume, ventilation, and distribution (Fig. 4*C*). The vital capacity was less than 70 per cent of the predicted value. Although bronchospirometry was not performed, inspection, auscultation, and fluoroscopy showed that the contribution of the right lung to respiration was grossly reduced. The physical signs and the normal index of intrapulmonary mixing indicated that the cyst communicated poorly, if at all. That proportion of the intrapleural volume occupied by the cyst could therefore not be measured, for the Darling technique is based on collecting gas ($N_2$) from air-containing space. Despite the presence of this large space-filling structure, the residual volume of the lungs was moderately elevated, indicating that the patient was unable to reduce the measurable volume of air in parenchyma to a normal minimum by expiring forcefully. Severe obstruction to air flow contributed to this inability. This was indicated by the progressively diminishing slope of the expiratory tracing (inscribed in the volumetric schema in Fig. 4*C*) and by the figures for the timed vital capacity. Failure of the mid-position to be promptly restored following an expiratory effort, shown in Fig. 4*C*, is characteristic of obstructive breathing. In keeping with these findings, the maximum breathing capacity was low.

Ventilation at rest was moderately increased to 5.5 L. per square meter of body surface area (normal 5 L.)—and was high in relation to oxygen uptake, the ventilatory equivalent being 3.2 (the normal range 2.2 to 2.6). In contrast to the previous patient, however, hyperventilation had not resulted in abnormally low arterial carbon dioxide tensions and contents (Table II), indicating that *alveolar* ventilation was proportional to carbon dioxide production.

Both dead space and venous admixture ratios were high, indicating an abnormal relationship between the distribution of gas and blood within the lungs. Since, as concluded from physical signs and mixing index, the cyst remained virtually unventilated, these distribution defects could be ascribed not to the cyst itself but either to the effect of the cyst on ipsilateral parenchyma or to abnormalities of the contralateral lung. Following resection, the arterial saturation at rest rose to normal values and the venous admixture component was restored nearly to normal. Coupled with the improved spirogram and timed vital capacity, these postoperative values suggested that the major portion of the preoperative abnormalities could be attributed to the effect of the cyst on adjacent parenchyma rather than to contralateral pulmonary pathology.

It should be observed, however, that the dead space ratio was unaltered by operation and that some abnormality of arterial blood oxygenation remained; the venous admixture ratio was moderately elevated and the saturation at exercise was abnormally low.

In addition to the favorable effect on oxygenation of arterial blood and on resistance to gas flow during expiration, excision of the cyst was followed by an increase in vital capacity (both inspiratory capacity and expiratory reserve) and total lung capacity and by a fall in the residual volume. Whereas the ratio of the residual volume to the total lung capacity had been abnormally high preoperatively, it fell to within normal limits postoperatively. The construction of a pleural tent contributed to the fall in residual volume and limited the restoration of the total lung capacity.

The remaining abnormality in maximum breathing capacity, and indeed in the other aspects of function already noted, may be ascribed in part, at least, to the brevity of the patient's postoperative convalescence.

*Summary*.—This patient, with a large unilateral air cyst, demonstrated severe abnormalities of ventilation, lung volumes, venous admixture, and physiologic dead space. Intrapulmonary mixing was normal. Together with the physical signs, the data were interpreted to indicate very poor communication of the cyst and gross interference with the function of ipsilateral lung. Following excision of the cyst the patient demonstrated more nearly normal lung volumes, reduced resistance to air flow within the airways, and improved oxygenation of arterial blood. The residual abnormalities in lung volumes, ventilation, and distribution were ascribed to incomplete recovery of function in lung which had re-expanded following a protracted period of compression.

### PATIENT NUMBER FIVE

*Clinical Summary*.—This 43-year-old white male writer first entered the hospital on May 20, 1954, with symptoms of one and one-half years' duration of slight to moderate dyspnea on exertion. There had been no orthopnea, cough, or hemoptysis. Pre-employment roentgenograms in January and March of this year had revealed the presence of bilateral pulmonary cysts.

The chest was symmetrical with equal expansion. There were hyperresonance and absent breath sounds in the left infraclavicular space. The extremities exhibited no clubbing or cyanosis.

X-rays of the chest showed two large areas of increased radiolucency involving the left upper lobe and a considerable portion of the right upper lobe. Bronchography revealed marked compression and displacement downward of the left upper lobe bronchi and crowding of the right upper lobe bronchi. Tomography (Fig. 5A) demonstrated a large cyst virtually replacing the left upper lobe and multiple cysts of the right upper lobe. Pulmonary function studies were performed (Fig. 5C).

On July 14, 1954, left thoracotomy was performed. There was a cyst 14 cm. in diameter occupying the apical posterior segment of the left upper lobe together with three adjacent cysts each approximately 5 cm. in diameter. Incision of the large cyst demonstrated patent connecting bronchi. The cysts were removed by a wedge technique. A pleural tent was created to tailor the remaining pleural space to the volume of the remaining lung parenchyma.

On return to the hospital in November of 1954 the patient had noticed subjective improvement in dyspnea and increased ability to do moderately strenuous work. On Nov. 10, 1954, a right thoracotomy revealed many large, thin-walled multilocular cysts of the apical and posterior segments of the right upper lobe. These were readily filled by positive pressure but deflated extremely slowly when suction was applied to the endotracheal tube. The remainder of the upper lobe contained fibrotic tissue and numerous [illegible] cysts

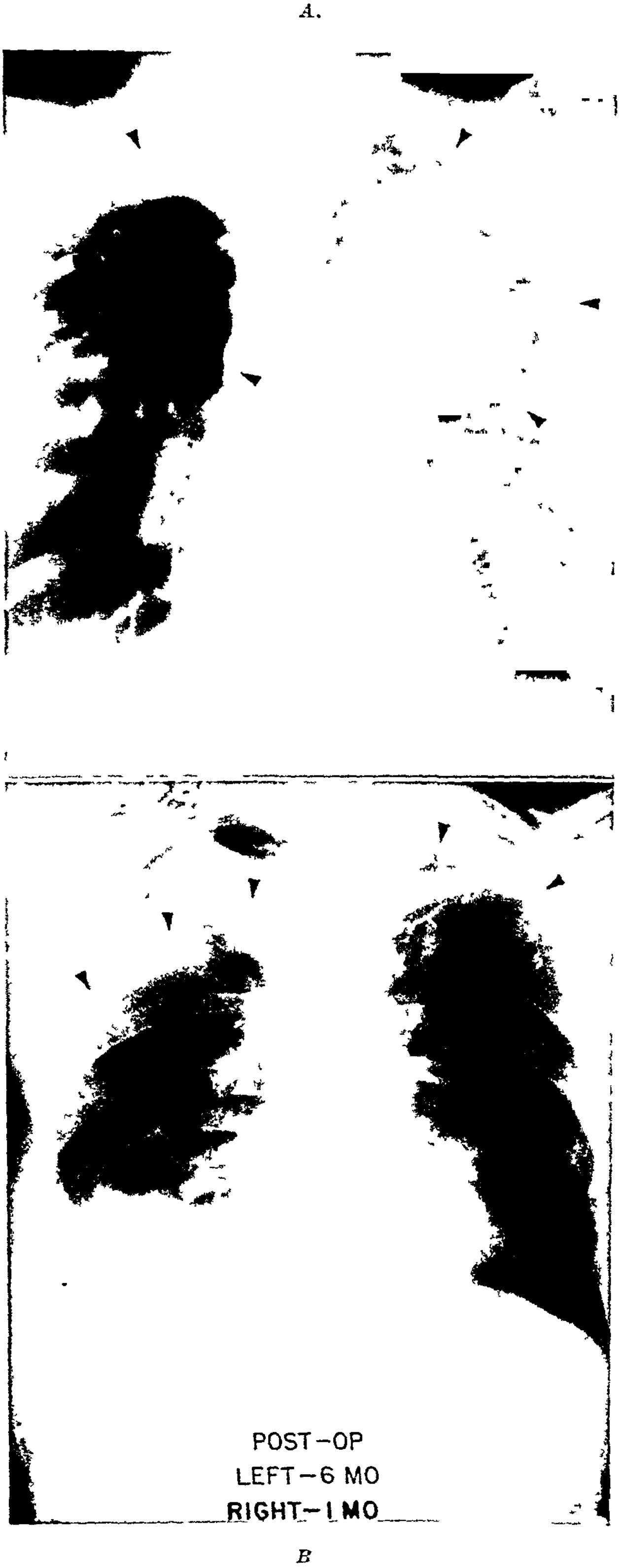


Fig. 5.—A, Preoperative tomogram showing bilateral apical radiolucent areas
B, Chest roentgenogram following completion of staged resection of bilateral lung cysts Arrows indicate pleural tents

A right upper lobectomy was accomplished and a pleural tent constructed (Fig. 5*B*). Lung function studies were reported before and after the second operation (Fig. 5*C*).

*Physiologic Studies.*—The preoperative values for this patient with extensive bilateral cystic disease were remarkably good The maximum breathing capacity, vital capacity, total lung capacity, and mixing index were normal or nearly so. The timed vital capacity, however, was definitely slowed, and the residual volume was elevated. There were, therefore, increased resistance to air flow on expirat'on and inability to reduce the volume of the lungs to a normal minimum by forceful expiration.

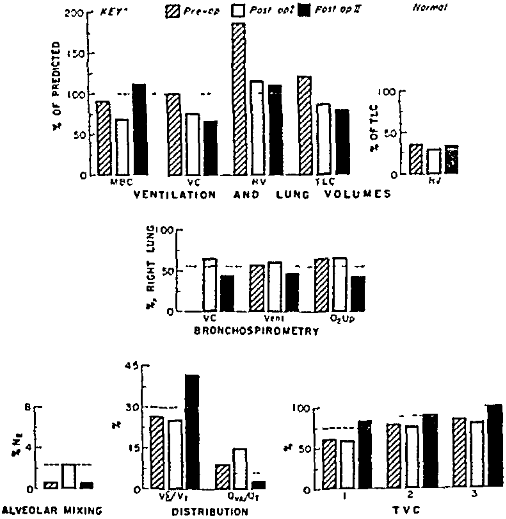


Fig 5*C*—Pre- and postoperative function data of Patient 5 In spite of bilateral cystic disease, tests of preoperative function did not disclose striking abnormalities See text for discussion

The fact that the index of intrapulmonary mixing was normal indicated that the cysts either did not communicate at all or communicated so completely as to be cleared of nitrogen during the period of oxygen breathing. In the latter instance one would expect the physiologic dead space to be abnormally high The normal figure for this measurement suggested extremely poor communication Slowing of the timed vital capacity, therefore, could probably not be ascribed to evacuation of large cystic areas through attenuated orifices This impression was confirmed at fluoroscopy by the finding that the dimensions of the cysts appeared to remain constant during deep breathing. The slowed timed vital capacity could then be attributed either to obstructive parenchymal disease exclusive of the cysts or, as was more likely in view of the good maximum breathing capacity, to a regional obstructive effect of the cysts on adjacent tissue. The improvement in maximum breathing capacity which followed excision of the cysts supported the latter explanation.

This patient, as well as the first 2, demonstrated that a poor timed vital capacity is compatible with a good maximum breathing capacity. By performing the latter test with an elevated position of the chest, some patients with obstructive pathology may ventilate well using the portion of their respiratory cycle characterized by the least obstruction.

In addition to improving ventilation (maximum breathing capacity), removal of the cysts produced changes in the lung volumes. The interpretation of these was colored by the space-filling procedures (pleural tents) concomitantly performed. The fall in vital capacity and total lung capacity could be attributed either to thoracotomy or to restriction of the endothoracic space. The striking fall in residual volume was probably a result of the latter, for similar changes do not usually follow uncomplicated bilateral resections.[18]

The results of bronchospirometry before and after the first operation were unusual in that the contribution of the left lung to ventilation and oxygen uptake was not adversely affected by ipsilateral pulmonary resection. This indicated that the tissue excised was relatively functionless and that the operation interfered minimally with the performance of remaining tissue. The second operation, however, reduced the contribution of the right lung to abnormally low levels. Probably, therefore, functioning tissue was excised together with the cyst. The operative note suggested that such tissue had been removed with the cyst.

Between the first and second operations, the index of alveolar mixing more than doubled its preoperative value in spite of the fact that a decrease might have been expected by virtue of the considerable fall in functional residual capacity which followed the first operation. Increased communication of the remaining cysts on the right side was a possible explanation for this change. An x-ray report dated one week following the second study described "less radiolucency at the right apex." This finding, together with reversion of the mixing index to preoperative levels after removal of the right upper lobe, suggested that communication had improved and that poor mixing in the cystic areas had indeed accounted for the high alveolar index.

The variations in distribution ratios are difficult to interpret. There was an increase in venous admixture following the first operation and, after the second operation, a pronounced rise in the ratio of the dead space to the tidal volume. The former indicated that after the first operation a larger proportion of the cardiac output was delivered to alveoli which were poorly ventilated relative to their perfusion. The dead space ratio is sometimes elevated out of proportion to an elevation in venous admixture in patients with emphysema.[19] A similar effect is produced when the $Pa_{CO_2}$ is overestimated due to error in its measurement. This determination was carried out in quadruplicate with good checks. In the absence of an analytical error, the reason for the unusual postoperative pattern of distribution in this patient remained unexplained.

Finally, it should be observed that the improvement in timed vital capacity (Fig 5C) reflected not an increase in the volume of air expelled in 1, 2, or 3 seconds but the considerable decrease in the vital capacity. Indeed, less air was moved postoperatively in each of the 3 time intervals than had been expelled preoperatively.

*Summary.*—This patient with bilateral cystic disease, in spite of respiratory symptoms, had a good maximum breathing capacity, nearly normal lung volumes, and minimal abnormalities in ventilation-perfusion relationships. The timed vital capacity was definitely slowed, however, and the mixing index was minimally elevated. Although it had been predicted preoperatively that the cysts on neither side communicated well, inspection of the pathologic specimens demonstrated anatomically patent communications on the left. Bilateral resections with construction of pleural tents were followed by an improved maximum breathing capacity, better intrapulmonary mixing, a fall in the major volumetric compartments and relief of subjective symptoms.

### PATIENT NUMBER SIX

*Clinical Summary.*—This 31 year old Negro man first noted the onset of pain in the right upper chest and progressive exertional dyspnea in March of 1953. Past history and systemic inquiry were noncontributory except for an episode of spontaneous pneumothorax of the left lung in 1951.

Physical examination disclosed diminished expansion of the chest. Tactile fremitus, vocal fremitus and breath sounds were absent from the right upper chest both anteriorly and posteriorly. There were râles at the right base, posteriorly. Roentgenographic examination of the chest revealed a large air containing cyst in the right lung (Fig 6A). Pulmonary function studies were obtained (Fig 6C).

At thoracotomy, on Aug 4, 1953, the pleural space was found to be completely obliterated by adhesions. It was impossible to collapse the lung because of the presence of innumerable large cysts in which the air was trapped. The cysts, varying from the size of a "football" to that of a "match head," were almost all located in the right upper lobe. They were removed by wedge resection.

Postoperative chest roentgenograms (Fig 6B) revealed one large and several smaller cysts in the left lung, these having become apparent following the contralateral surgery. Further pulmonary function studies were obtained (Fig 6D).

On Sept 24, 1953 a left thoracotomy disclosed a large thin walled, air containing cyst which immediately ballooned out through the incision. This cyst measuring 12 by 8 cm, was attached to the anterolateral edge of the lingula. There were also three "lemon sized" cysts arising from the midanterior portion of the left upper lobe and multiple smaller cysts and emphysematous blebs scattered throughout the remaining parenchyma. The left lo [illegible] was grossly normal except for the presence of small emphysemator

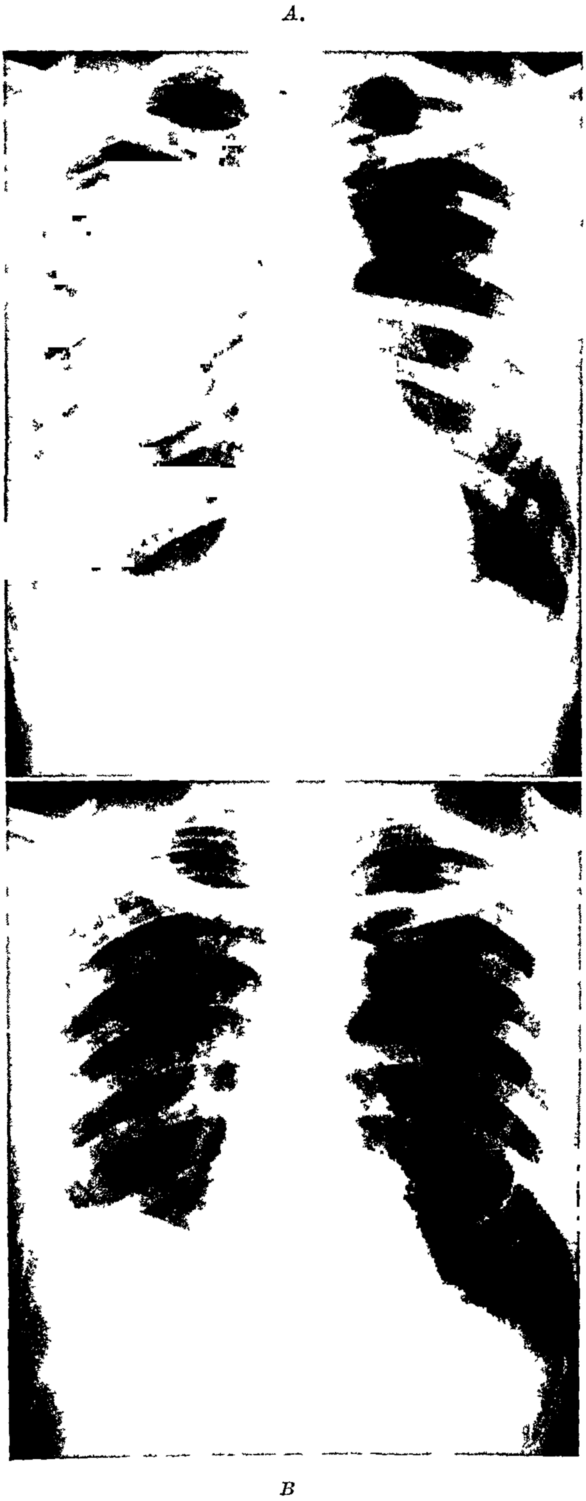


Fig. 6.—*A*, Chest roentgenogram Note large radiolucent area occupying the upper half of the right hemithorax.

*B*, Chest roentgenogram. Right lung cyst has been excised Note the appearance of the radiolucent area in the left hemithorax.

larger cysts were removed by wedge resection with the pleura being approximated over the exposed lung tissue. Large bronchi were encountered in the resection of the cysts.

The postoperative course was uneventful. Following convalescence after the second thoracotomy, the patient was capable of moderate exertion without feeling short of breath.

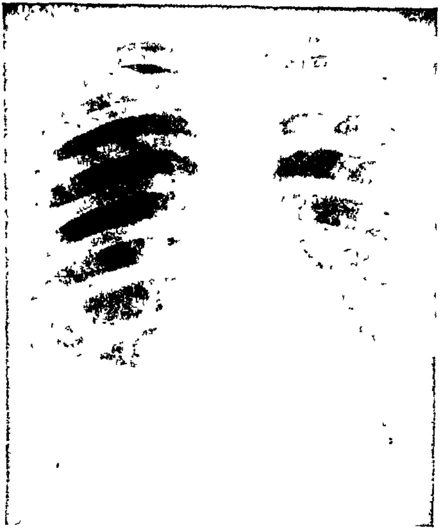


Fig. 6C.—Chest roentgenogram Appearance of chest following bilateral excision of lung cysts

*Physiologic Studies.*—In contrast to the previous patient with bilateral cystic disease, the present one demonstrated a number of obvious respiratory abnormalities. The maximum breathing capacity, vital capacity, and total lung capacity were reduced. The contribution of the right lung to function was abnormally low (less than 35 per cent of total). The mixing index was moderately elevated. The venous admixture was extremely high and the maximum diffusing capacity was very low.

Postoperatively, the values for all tests were more nearly normal. Ventilation, lung volumes, intrapulmonary mixing, arterial blood gas contents, and dimensions of the alveolar-capillary bed (maximum diffusing capacity) were all favorably affected. Therefore, a major portion of the preoperative respiratory pathology could be ascribed to the effects of the cysts rather than to other parenchymal disease. Preoperatively most of the right lung was compressed into the right lower chest ("vanishing lung syndrome") and could not normally circulate either air or blood. Ventilation and mixing were therefore poor and the measurable lung volumes were small. Perfusion remained more nearly normal than ventilation with the result that an unusually large proportion of the blood flowing through the lungs was poorly oxygenated.

Saturation of peripheral arterial blood with oxygen was therefore low (92 per cent at rest, 88 per cent during steady-state treadmill exercise), and the venous admixture ratio was unusually high (24 per cent). Carbon dioxide content (46.92 vol. per cent) and tension (35 mm. Hg), however, were low in contrast to what one might expect in a mixture containing 24 parts of mixed venous blood and 76 parts of well-arterialized blood. The explanation for this

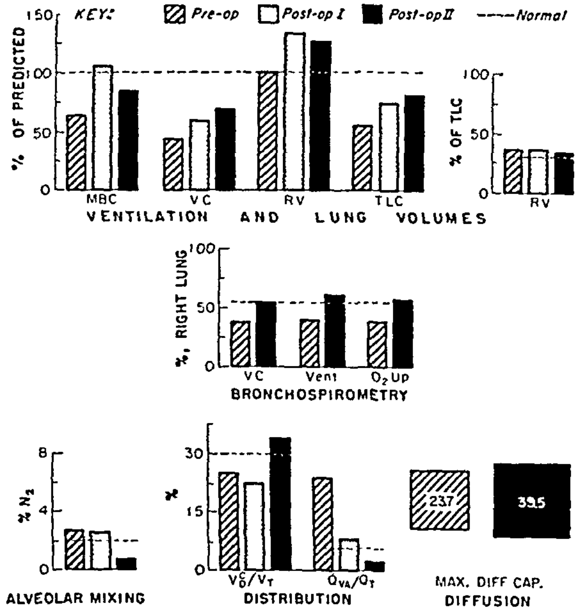


Fig. 6D.—Pre- and postoperative function data of Patient 6. Preoperatively there were severe abnormalities of ventilation, lung volumes, relative performance of the two lungs, ventilation-perfusion relationships, and the maximum diffusing capacity. With the exception of the physiologic dead space, all aspects of function were favorably affected by operation.

apparent physiologic discrepancy can be visualized by reference to the differences in the dissociation curves for carbon dioxide and oxygen. These curves show that although it is not possible to augment the arterial oxygen content by regional hyperventilation (flat part of $O_2$ dissociation curve where increases in $P_{O_2}$ result in only small changes in $O_2$ content), lowering alveolar $P_{CO_2}$ by this mechanism will cause a loss of $CO_2$ from the blood. Regional hyperventilation, although ineffective in compensating for poor oxygenation due to localized hypoventilation, is more nearly effective in maintaining normal blood levels of carbon dioxide.

Saturation of peripheral arterial blood is maintained in part by constriction of pulmonary vessels in response to hypoxia.[20] It is evident that, in this patient, this mechanism for shunting blood away from the hypoventilated areas was incompletely effective.

The low diffusing capacity together with the postoperative rise in this measurement suggested that a number of alveolar-capillary units were ineffectual as a result of the cysts' pressures. It should be noted that even postoperatively the value for this test was less than normal. This could be attributed to excision of functioning alveoli contiguous to the cysts, to incomplete postoperative recovery of function, and to the presence of additional small blebs representing destruction of alveoli. The latter were described in the operative note.

Most of the functional improvement was produced by the first operation and may be accounted for by re-expansion of compressed lung. The residual volume was increased because the contents of the entire right hemithorax now contributed to the functional residual capacity; preoperatively, a large proportion of the intrapleural volume had been filled by the noncommunicating air-filled cysts. The maximum diffusing capacities after the first and second operations were nearly identical, indicating that the tissue excised from the left lung at the second operation included a minimum number of functioning alveoli. As was true of the diffusing capacity, persistence of low values for tests of ventilation and lung volumes reflected in part the effect of the short convalescence. Finally, the poor communication suggested preoperatively by the mixing indices was confirmed at thoracotomy.

*Summary.*—This patient with bilateral air cysts and severe respiratory abnormalities improved strikingly after excision of the cysts permitted re-expansion of compressed lung. The improvement was measured in terms of ventilation, lung volumes, intrapulmonary mixing, ventilation-perfusion relationships, and the total maximal area of the effective alveolar-capillary bed. The preoperative prediction that the cysts on both sides communicated poorly was borne out at thoracotomy. The respiratory deficits remaining postoperatively were attributed to both the brevity of the patient's convalescence and the remaining pulmonary pathology.

## DISCUSSION

The six studies of pulmonary cystic disease included in this report were selected (1) to serve as interesting exercises in the interpretation of pulmonary function tests, (2) to demonstrate the variability in the physiologic deficit associated with cystic disease of the lung, and (3) to document the beneficial effects of excisional therapy.

The first objective was approached by illustrating and discussing the patients' performance of certain pulmonary function tests.

With reference to the second and third objectives, these studies show that no single pattern of respiratory abnormality is characteristic of pulmonary cystic disease. Large cysts may be associated with a minimal, moderate, or extreme physiologic deficit incorporating not only abnormalities of ventilation and lung volume but of gas exchange as well.

In the absence of generalized pulmonary disease, the degree of the physiologic abnormality produced by a cyst is largely a function of the extent to which the cyst compresses the surrounding lung. This compression in turn is greater as the pressure difference between the inside of the cyst and the adjacent tissue increases. When a cyst communicates poorly or intermittently (Patients Nos. 4 and 6), this pressure difference remains high. Particularly on forced expiration, the cyst remains inflated at a high intracystic pressure at a time when the adjacent parenchyma contains its residual volume at oral or atmospheric pressure. The patency of communication, therefore, is a critical factor in the production of parenchymal compression. Accordingly, the index of alveolar mixing, an approximate measure of the degree of communication, is of particular significance.

As would be expected from the foregoing, those patients with the most severe physiologic deficits had, as judged from normal or minimally elevated alveolar indexes, poor or intermittent communication. In these, the effect of the cyst's pressure was not only to obstruct the flow of air to and from adjacent tissue but, in addition, to disrupt the normal relationship between ventilation and perfusion of this tissue. In Patient No. 6, for example, pre- and postoperative measurements of the maximum diffusing capacity showed that the number of alveoli available for the diffusion of oxygen into the blood stream was in effect diminished. These derangements together with abnormal ventilation-perfusion relationships and an attenuated diffusing surface are those one would expect in a lung in which partial and complete atelectasis were mixed. They fit the interpretations of roentgenograms and the descriptions of the lung at thoracotomy.

None of the patients had physiologically compromising generalized pulmonary emphysema. Two (Nos. 4 and 6), however, demonstrated hypoxia (excessively high venous admixture ratios) both at rest and exercise. In both instances, the saturation of arterial blood at rest returned to normal postoperatively and, in one instance (No. 6), to normal levels even on severe treadmill exercise. Poor saturation of arterial blood per se, therefore, was not an absolute contraindication to surgery. This point is stressed because of the emphasis it is given by Baldwin and her colleagues.[1] Among the 16 patients reported by these authors, hypoxia was found only in patients in whom cystic disease was superimposed on advanced pulmonary emphysema. Hypoxia under this circumstance is undoubtedly an adverse prognostic index. It is evident from the present patients that hypoxia may appear independently of emphysema. One would have excluded from surgery those patients who were to gain the most from it, had poor saturation of arterial blood in itself been interpreted to contraindicate resection.

The patient with far-advanced emphysema is, of course, a poor surgical risk and should be distinguished from one whose respiratory deficits can be ascribed primarily to cysts and their effects on relatively normal adjacent parenchyma. This distinction may be difficult, for the physiologic characteristics of each disease have much in common. However, in the 2 patients (Nos. 4 and 6) whose poor saturation of arterial blood might have led one to suspect advanced emphysema, other findings did not confirm this impression. The alveolar index was not high; the ratio of the residual volume to the total lung capacity was not unusually elevated, and there was no carbon dioxide retention. Physical findings and roentgenography, of course, are also of additional help in establishing the presence of generalized emphysema.

The correspondence between preoperative physiologic evidence of communication and the ease with which the cyst could be deflated at operation was good. The difference between physiologic and anatomic communication was demonstrated by Patient No. 3 whose specimen showed a number of patent airways despite evidence of poor functional communication. A valve mechanism at the orifices within the cyst is a possible explanation for this apparent inconsistency.

It is our opinion that the surgical treatment of few pulmonary lesions offers a more promising functional result than that demonstrated by these patients. As with a constricting pleural peel, the pulmonary cyst is largely functionless and the compressed adjacent tissue may be relatively normal. Optimal results may be expected when treatment is instituted early, for it is reasonable to assume that recovery of parenchymal function is adversely affected by duration and extent of compression.

## SUMMARY

The physiologic data of 6 patients with cystic disease of the lung were selected for presentation. The effects of surgical management were studied in 3 of these patients, 2 of whom had bilateral resections. Measurements of ventilation-perfusion relationships, of the maximum diffusing capacity, and of the total capacity of each lung individually were used to supplement the customary bilateral and bronchospirometric determinations of ventilation, lung volume, or oxygen uptake.

Four of the patients fell into recognized functional types: minimal or moderate abnormalities of ventilation and lung volumes with or without evidence of communication between the cyst and airways. Two patients presented patterns of physiologic abnormalities not previously described: severe abnormalities of ventilation, lung volumes, and arterial blood gas contents unassociated with generalized pulmonary emphysema.

In the absence of generalized emphysema, the patency of communication and the degree of parenchymal compression produced by the cysts appeared to be the most likely factors accounting for variation in the physiologic deficit. The preoperative impression of the extent of *communication and compression* correlated well with the anatomic findings.

Resection of the cysts produced beneficial symptomatic and physiologic effects. In the most severely ill patients, dyspnea was relieved, ventilation improved, lung volumes were restored toward normal, hypoxia was eliminated, and the maximal dimensions of the effective alveolar capillary bed were augmented.

Patient No. 6 was operated upon by Dr. Melvin Newman whose cooperation in referring the patient for study is gratefully acknowledged.

The clinical analyses, lung volumes, and tests of ventilation were performed by hospital corpsmen of the United States Navy trained in the Cardiopulmonary Laboratory of the United States Naval Hospital, St. Albans, New York. The authors are particularly indebted to the following for their skill and interest: Jerry Borrelli, Henry Haddad, Joseph Malin, John O'Brien, Robert Obser, Ernest Picknally, and Robert Rauf.

## REFERENCES

1. Baldwin, E., Harden, K. A., Greene, D. G., Cournand, A., and Richards, D. W., Jr.: Pulmonary Insufficiency. IV. A Study of 16 Cases of Large Pulmonary Air Cysts or Bullae, Medicine **29**: 169-194, 1950.
2. Warring, F. C., Jr., and Lindskog, G. E.: Surgical Management of Giant Air Cysts of the Lungs. Physiological Improvement After Resection, Am. Rev. Tuberc. **63**: 579-586, 1951.
3. Kaltreider, N. L., and Fray, W. W.: Pathological Physiology of Pulmonary Cysts and Emphysematous Bullae, Am. J. Med. Sc. **197**: 62-77, 1939.
4. Darling, R. C., Cournand, A., and Richards, D. W., Jr.: Studies on Intrapulmonary Mixture of Gases: Open Circuit Method for Measuring Residual Air, J. Clin. Invest. **19**: 609-618, 1940.
5. Siebens, A. A., Newman, M. M., Smith, R. E., and Vaughan, L. H.: Measurements of the Unilateral Residual Volume, J. THORACIC SURG. **31**: 569, 1956.
6. Van Slyke, D. D., and Neill, J. M.: The Determination of Gases in Blood and Other Solutions by Vacuum Extraction and Manometric Measurement, J. Biol. Chem. **61**: 523-525, 1924.
7. Riley, R. L., Proemmel, D. D., and Franke, R. E.: A Direct Method for Determination of Oxygen and Carbon Dioxide Tensions in Blood, J. Biol. Chem. **161**: 621-633, 1945.
8. Brinkman, G. L., Johns, C. J., Danoso, H., and Riley, R. L.: A Modification of the Method of Riley, Proemmel and Franke for Determination of Oxygen and Carbon Dioxide Tension in Blood, J. App. Physiol. **7**: 340-346, 1954.
9. Scholander, P. F.: Analyzer for Accurate Estimation of Respiratory Gases in One-half Cubic Centimeter Samples. J. Biol. Chem. **167**: 1, 1947.
10. Lilienthal, J. L., Jr., Riley, R. L., Proemmel, D. D., and Franke, R. E.: An Experimental Analysis in Man of the Oxygen Pressure Gradient From Alveolar Air to Arterial Blood During Rest and Exercise at Sea Level and at Altitude, Am. J. Physiol. **147**: 199-216, 1946.
11. Riley, R. L., and Cournand, A.: Analysis of Factors Affecting Partial Pressures of Oxygen and $CO_2$ in Gas and Blood of Lungs: Theory, J. App. Physiol. **4**: 77-101, 1951.
12. Riley, R. L., Cournand, A., and Donald, K. W.: Analysis of Factors Affecting Partial Pressures of Oxygen and Carbon Dioxide in Gas and Blood of Lungs. Methods, J. App. Physiol. **4**: 102-120, 1951.
13. Riley, R. L., Shepard, R. H., Cohn, J. E., Carroll, D. G., and Armstrong, B. W.: Maximal Diffusing Capacity of the Lungs, J. App. Physiol. **6**: 573-587, 1954.
14. Siebens, A. A., and Frank, N. R.: Diffusing Capacity at Exercise in Patients With Alterations in Area of the Pulmonary Capillary Bed, Am. J. Physiol. **179**: 673, 1954.
15. Riley, R. L., Riley, M. C., and Hill, H. McD.: Diffuse Pulmonary Sarcoidosis: Diffusing Capacity During Exercise and Other Lung Function Studies in Relation to ACTH Therapy, Johns Hopkins Hosp. Bull. **91**: 345-370, 1952.
16. Papenheimer, J. R.: Standardization of Definitions and Symbols in Respiratory Physiology, Fed. Proc. **9**: 602-605, 1950.
17. Klopstock, R., and Grant, A. R.: Discussion of Newman, Huggin, Butler, and Bowman; Surgical Treatment in Tuberculosis Complicated With Pulmonary Emphysema, J. THORACIC SURG. **31**: 125, 1956.
18. Smith, G. A., Siebens, A. A., and Storey, C. F.: Preoperative and Postoperative Cardiopulmonary Function Studies in Patients With Bronchiectasis, Am. Rev. Tuberc. **69**: 869-914, 1954.
19. Riley, R. L., and Cournand, A.: "Ideal" Alveolar Air and the Analysis of Ventilation-Perfusion Relationships in the Lungs, J. App. Physiol. **1**: 825-847, 1949.
20. Siebens, A. A., Smith, R. E., and Storey, C. F.: Effect of Hypoxia on Pulmonary Vessels in Man, Am. J. Physiol. **180**: 428-432, 1955.

# CHRONIC (HEALED) DISSECTING ANEURYSMS

John T. Prior, M.D., Robert T. Burns, M.D.,* and Theodore Peri, M.D.
Syracuse, N. Y.

DISSECTION of the aorta has come to be characteristically associated with the sudden onset of tearing pain, frequently radiating, followed by loss of consciousness, prostration, shock, coma, and death. This dramatic clinical concept of dissecting aneurysms, referring as it does to the acute variety, only tends toward obscuring the fact that a small proportion of this type of aneurysm attempts a healing response to this poorly understood type of mural arterial alteration.

A review of a large number of cases of dissecting aneurysm which have come to autopsy at this Center disclosed five examples of the so-called "healed" variety. It is the purpose of this report to present the clinical and pathologic manifestations of these cases in detail. We feel that the pathologic changes within the aorta, of this series, exhibit the wide range of reparative phenomena that is possible, extending from imperfectly organized intramural hematomata to long-standing secondary endothelial-lined channels with mural elastification and intimal hyperplastic changes. Additional interest obtains in cases of this type by reason of the great advances in the field of cardiovascular surgery, several reports of surgical repair of dissecting aneurysm being found in the recent literature.[1-4]

Some of the data relevant to these cases are outlined in Table I.

TABLE I. SUMMARY OF CASES

| CASE NUMBER | AGE | SEX | DURATION LIFE | HEART WEIGHT | TERMINAL BLOOD PRESSURE | CAUSE OF DEATH |
|---|---|---|---|---|---|---|
| 1—R. I.<br>(A55-20) | 87 | M | >6 mo. | 780 Gm. | 118/88 | Acute dissection of aorta into pericardial space |
| 2—A. M.<br>(UA40-24) | 38 | F | 1 year | 600 Gm. | LA—260/150<br>RA—200/146 | Chronic rheumatic heart disease with failure |
| 3—A. P.<br>(A55-29) | 72 | M | 7½ mo. | 515 Gm. | 180/130 | Congestive heart failure |
| 4—J. L.<br>(A48-199) | 43 | M | 63 days | LV—20 mm.<br>RV—6 mm. | 180/110 | Aortic rupture into left pleural space |
| 5—A. J.<br>(A53-229) | 86 | F | Years | 345 Gm. | 220/110 | Carcinoma of the stomach with metastases |

---

From the Departments of Pathology and Radiology, State University of New York Upstate Medical Center, Syracuse, N. Y.

Received for publication Jan. 3, 1956.

*Present address: Strong Memorial Hospital, Rochester.

## CASE REPORTS

CASE 1.—R. L. (A55-20), an 87-year-old white man, was apparently well until approximately Jan. 23, 1955, when a diagnosis of coronary artery occlusion was made. He was not hospitalized and it is not known whether an electrocardiogram was obtained.

Two weeks later he was admitted to Syracuse Memorial Hospital complaining of substernal pain, weakness, and a cold-sweating feeling. An accurate past history could not be obtained. Physical examination disclosed an elderly man who was extremely pale, clammy, and in acute distress. Respiratory distress was present. The temperature was 97° F., pulse 126 per minute, and the respirations were 30 to 32 per minute. An 8 by 10 cm. soft, mobile mass was noted in the left neck which pushed the trachea to the right of the midline but did not pulsate. Dullness to percussion was described in the left axilla and base of the left chest and râles were heard at both lung bases. The left border of cardiac dullness was found to be in the left midaxillary line. The heart sounds were of poor quality and a tic-toc rhythm, with some irregularities, was noted. A Grade II precordial systolic murmur and a questionable diastolic murmur were heard in the left axilla. The abdomen was soft and the liver edge could be outlined 1 to 2 fingerbreadths below the right costal margin. The lower extremities were not remarkable. Laboratory data revealed a hemoglobin of 12.5 Gm. per cent; hematocrit, 36 per cent; and a total white blood cell count of 25,000 per c.mm., wth 38 per cent polymorphonuclear leukocytes and 21 per cent lymphocytes. Electrocardiographic changes were interpreted as acute ischemia and injury to the anterolateral septal area with subendocardial ischemia. The patient died on the day of admission.

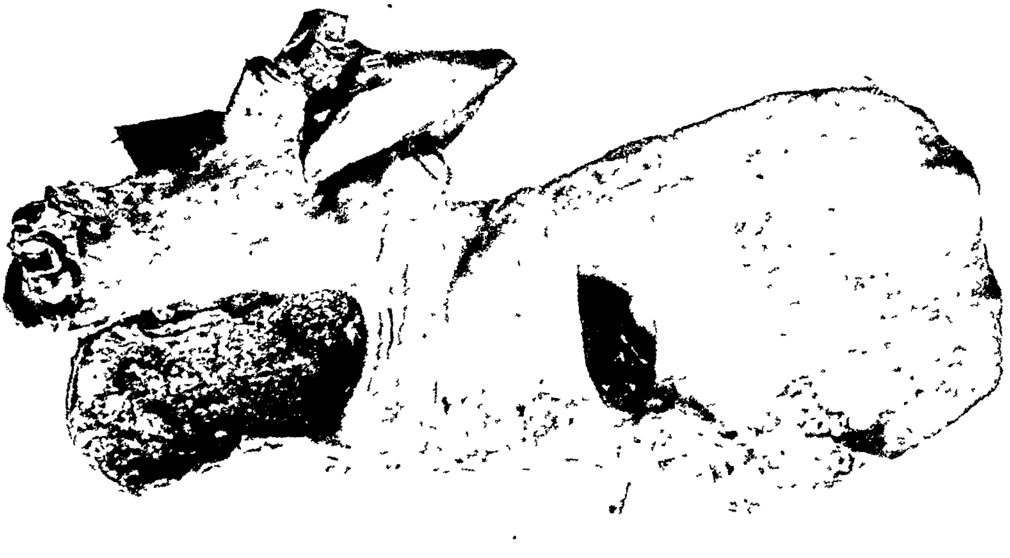


Fig. 1.—Case 1. Aortic intimal surface showing the two clot-filled defects, the larger being more proximal. The central intimal zone to the right of the distal defect is free of atherosclerosis.

At autopsy the principal findings were confined to the heart and the aorta. The heart weighed 780 Gm. due to a left ventricular thickness which was twice normal. The valves were not unusual but there was a fibrotic area in the posterior left ventricle consistent with an old myocardial infarction. The aorta was the site of a recent and an old dissection. The old dissection was located in the thoracic aorta while the recent one was in the ascending portion of the arch with rupture into the pericardium and death was due to cardiac tamponade This recent dissection was not contiguous with the older process and there was no evidence of an intimal tear The recent dissection involved the innominate artery but the left common carotid and left subclavian arteries were spared. The healed aneurysm was located in the proximal descending aorta and presented an hour glass shaped defect which began just below the origin of left subclavian artery This deformity was oriented in the longitudinal plane of the aorta, measured 15 by 4 5 cm, and appeared to involve 180 degrees of the posterolateral aortic circumference Viewed from its intimal surface, there were two large defects present (Fig 1) The more cephalad defect was a shell shaped 7 by 4 cm depression filled with impacted blood clot The second defect was oval in shape, measured 3 5 by 2 cm, and was located 5 5 cm distal to the lower border of the proximal rent. A sagittal section of this entire area disclosed that although there had once been continuity of intramural hematoma between these tears, it was now nearly obliterated by dense fibrotic tissue which produced the constriction in the hourglass deformity (Fig. 2). The clot within the more caudad depression was easily removed and there appeared to be a nearly complete endothelial lining about this clot Examination of Fig 1 gives the impression that the new endothelium was continuous with a pearly white longitudinal zone that extended distally for a distance of 12 cm

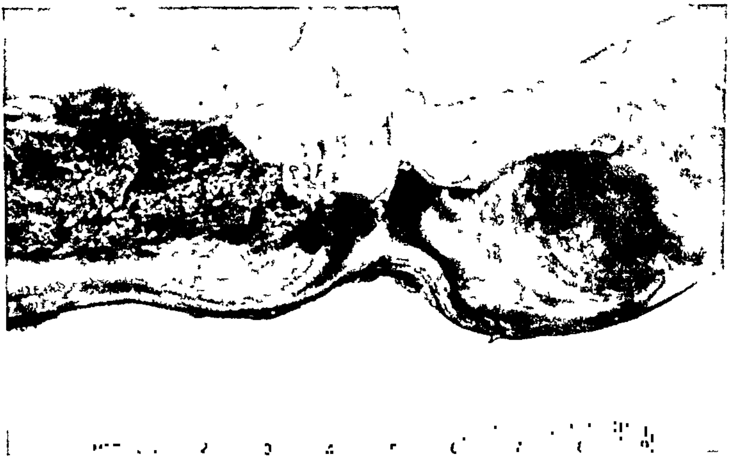


Fig 2—Case 1 Sagittal section illustrating the central organization producing the hour glass deformity

Microscopic examination of the area of recent dissection showed very slight evidence of Erdheim's cystic medionecrosis Minimal atherosclerosis was present Study of the old dissection disclosed fibrosis of the entire thickness of the aortic wall in the vicinity of the proximal tear The intramural hematoma was composed of amorphous eosin staining material without evidence of significant organization or cellular debris The vasa vasorum were thickened and there was a prominent lymphoid infiltrate present The most narrowed point shown in Fig 2 was composed of dense connective tissue with many prominent large vessels and the overlying intima at this point was the site of marked atherosclerosis The aortic wall below the distal defect showed mural fibrosis in its outer third only Of considerable interest was the fact that the most severe cystic me[illegible] located beneath the intima described previously as a pearly white longi[illegible]tal to the more caudad intimal tear (Fig. 3). This area was unusually striking in the gross examination since it was virtually free from atherosclerosis although this process was quite severe along its lateral limits. Since this dissection could not be accurately dated, its duration (more than 6 months) was estimated by comparing the microscopic alterations in this case with others in which the original date of dissection was known.

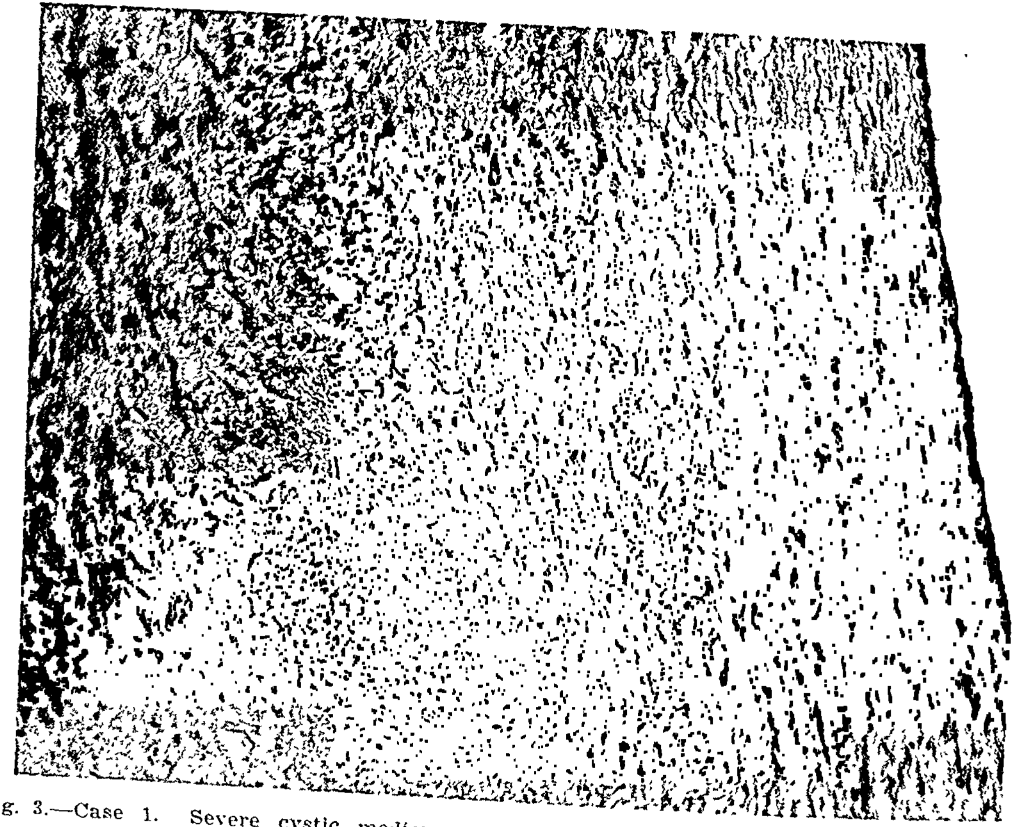


Fig. 3.—Case 1. Severe cystic medionecrosis in the atherosclerotic-free intima shown in Fig. 1. (×100.)

CASE 2.—A. M. (UA40-24), a 38-year-old white woman, entered Syracuse University Hospital of the Good Shepherd April 9, 1940, complaining of weakness, *anorexia*, nervousness, intermittent precordial pain, a cough of 6 months' duration, and some swelling of the extremities. She was said to have had coronary artery thrombosis one year previously, with an accompanying fall in blood pressure. Since that time she had noticed occasional sharp precordial pain with radiation to the left arm.

Physical examination disclosed a blood pressure in the left arm of 260/150 mm. Hg and 200/146 mm. Hg in the right arm. The patient was dehydrated, cyanotic, and responded very slowly. The pupils reacted porly to both light and accommodation and the left pupil was larger than the right. Venous congestion was apparent. The heart was enlarged both to the right and to the left as determined by percussion. Loud presystolic, systolic, and diastolic murmurs were heard over the mitral area and a systolic murmur was noted over the tricuspid area. The sounds were of poor quality and the rhythm was totally irregular. A few râles were heard at the base of the left lung, and the liver was located 5 fingerbreadths below the right costal margin and was described as tender and pulsating. Deep tendon reflexes were more active on the right.

Laboratory data disclosed the Wassermann reaction to be negative. The hemoglobin was 11 Gm. per cent; red blood cell count 3.8 million per c.mm., white blood cell count was 38,400 per c.mm., with 96 per cent polymorphonuclear leukocytes, 3 per cent lymphocytes, and 1 per cent monocytes. The urine examination disclosed it to be acid, with a specific gravity of 1 022 a 4 plus albumin and clumps of both red and white blood cells were seen on microscopic examination The nonprotein nitrogen was 125 mg per 100 c c The electrocardiogram was interpreted as showing left ventricular preponderance auricular fibrillation, and myocardial damage Roentgenographic examination of the chest disclosed the greatest diameter of the chest to be 26 cm the greatest diameter of the heart to be 20 cm and there was marked widening of the heart shadow due to left ventricular hypertrophy The left auricular appendage was not prominent The trachea deviated to the right posterior and there was some fullness of the supracardiac shadow There was increased density lateral to the aortic shadow and above each root These areas lateral to the aortic shadow were thought to be due to congestion, inflammation or possibly tumor The patient died on the third day after admission

At autopsy, there was evidence of acute and chronic rheumatic heart disease, mitral stenosis, a mural thrombus in the left auricular appendages, left ventricular hypertrophy (heart weight—600 Gm ) chronic passive congestion of the liver arteriolar nephrosclerosis bronchopneumonia, and focal glomerulonephritis Permission for examination of the brain was not obtained Examination of the aorta disclosed it to be enormously widened from the distal end of the arch to the iliac bifurcation Inspection of the aortic interior revealed the presence of two distinct lumens at the distal end of the aortic arch leading into a so called "double barrel" aortic structure (Fig 4) The larger lumen was continuous with an intramural secondary channel which had been produced by a previous aortic dissection This secondary passage extended down the length of the aorta to terminate in the left iliac artery where it recommunicated with the original vascular system The lining of this secondary channel was light yellow somewhat wrinkled, and contained organized intimal thrombi which extended from its origin to a point just below the celiac axis Moderate atherosclerosis was present within the true lumen

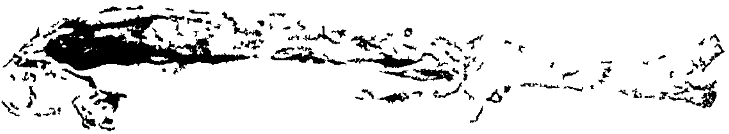


Fig 4 —Case 2 Double barrel aorta with both channels only partially opened The false channel contains organized blood clot

Microscopic examination of the true aortic wall disclosed a moderate amount of cystic medionecrosis within the ascending aorta and aortic arch while the process was virtually absent within the thoracic and abdominal aorta Atherosclerosis was diffusely present along the entire length of the true aortic intima The secondary channel was characterized by a thick wall, a new endothelial lining, and by marked subintimal proliferation With the use of Verhoeff's elastic stain a large number of new elastic fibers were noted to have developed within the wall of the secondary channel (Fig 5) The most striking abnormality, however, was the large number of focal, eccentrically located zones of intimal thickening Although devoid of elastic tissue, they were composed of an embryonic type of loose mesenchymatous tissue very similar to that seen in Wharton's jelly of the umbilical cord (Fig 6) The fibroblasts within these subintimal thickenings were stellate shaped and there was a large amount of pale homogeneous intercellular substance which appeared almost fluid in nature Although several observers have described the presence of arteriosclerosis within the secondary channel,[5-7] we failed to see any similarity between this type of intimal change and any variant of human arteriosclerosis It did, however, *appear to be identical with the changes described by Prior and Jones*,[8] who noted

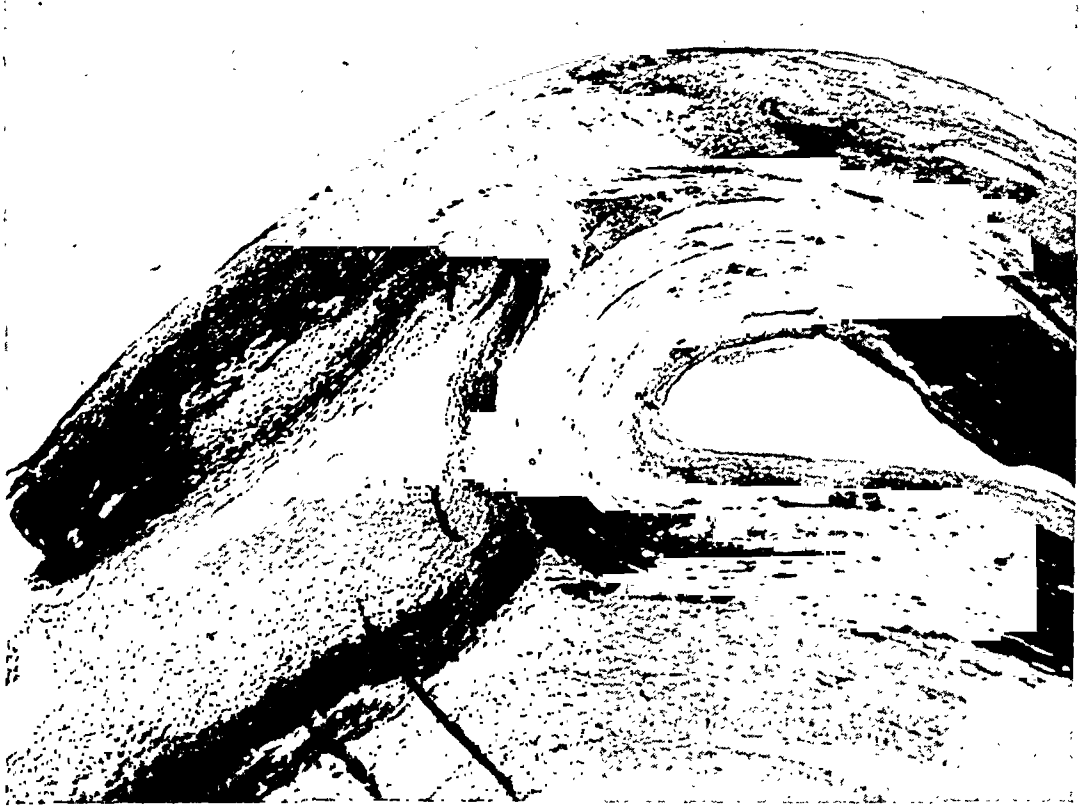


Fig. 5.—Case 2. Verhoeff's elastic tissue stain showing new elastic tissue *formation* in the secondary lumen toward the left side of the illustration. The new intimal connective tissue in this channel can be seen. (×20.)

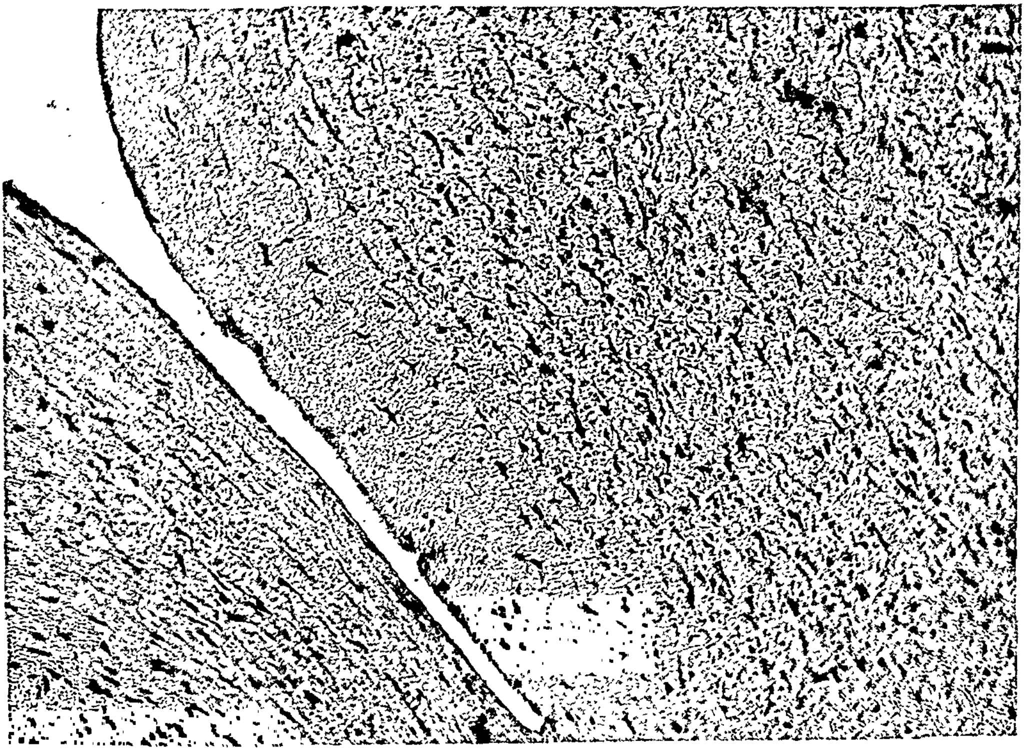


Fig. 6.—Case 2. Higher-power magnification of the newly formed intimal and subintimal connective tissue within the secondary channel. (×65.)

persistence of mesenchyme-like structure within the aortic intima and within the intima and media of the great vessels arising from the arch at birth, and for a considerable length of time thereafter in the newborn infant.

Case 3.—*A. P. (A55-29), a 72-year-old white man, was well until June 19, 1954, when* he complained of continuous subxiphoid pain. The first pain appeared suddenly and while he *was at rest. Additional complaints of exertional dyspnea, paroxysmal nocturnal* dyspnea and two-pillow orthopnea were elicited by the examiner. The pain disappeared on July 12, 1954, and hypertension was first noted one month later. A roentgenogram of the chest taken Jan. 5, 1953, prior to the onset of symptoms, was interpreted as showing slight mediastinal widening. A subsequent roentgenogram taken on Aug. 13, 1954, after the onset of symptoms, showed enlargement of the heart and a dilated tortuous aorta. No pleural fluid was seen but there was some pulmonary vascular congestion indicating heart failure.

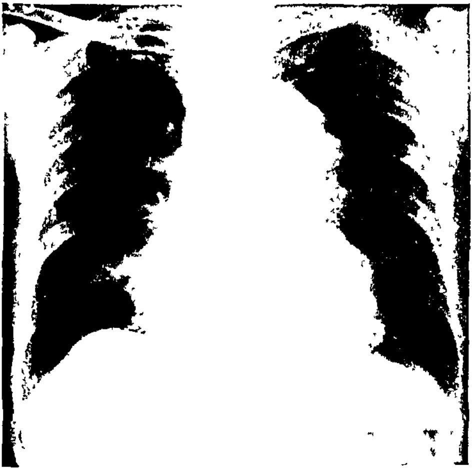


Fig. 7.—Case 3. Chest roentgenogram showing the peculiar segmental flattened appearance of the convex aneurysmal surface.

Six weeks later, on Sept. 27, 1954, this patient was admitted to Syracuse University Hospital of the Good Shepherd complaining of shortness of breath and inability to sleep *for the preceding week. The symptoms of dyspnea, orthopnea, and paroxysmal nocturnal* dyspnea persisted. He was digitalized at this time and maintained on 0.1 Gm. of digitalis leaf daily. The blood pressure on admission was 200/134 mm. Hg. Diffuse inspiratory coarse râles were heard over both lung bases. The heart was slightly enlarged to percussion and the liver was 2 fingerbreadths below the right costal margin in the midclavicular line. No mention of an absent pulse in any extremity was made. A roentgenogram of the chest and fluoroscopic studies showed a dilated tortuous aorta at this time (Fig. 7). The Wassermann reaction was negative and other laboratory data were noncontributory. An electrocardiographic examination was *interpreted as showing left ventricular enlargement.* The patient was discharged Oct. 29, 1954, without signs of cardiac failure.

Additional chest roentgenograms were taken in the outpatient department on Dec. 30, 1954, approximately six and one-half months after the onset of symptoms. The heart remained slightly enlarged and the lungs were clear. There had bc [illegible] further

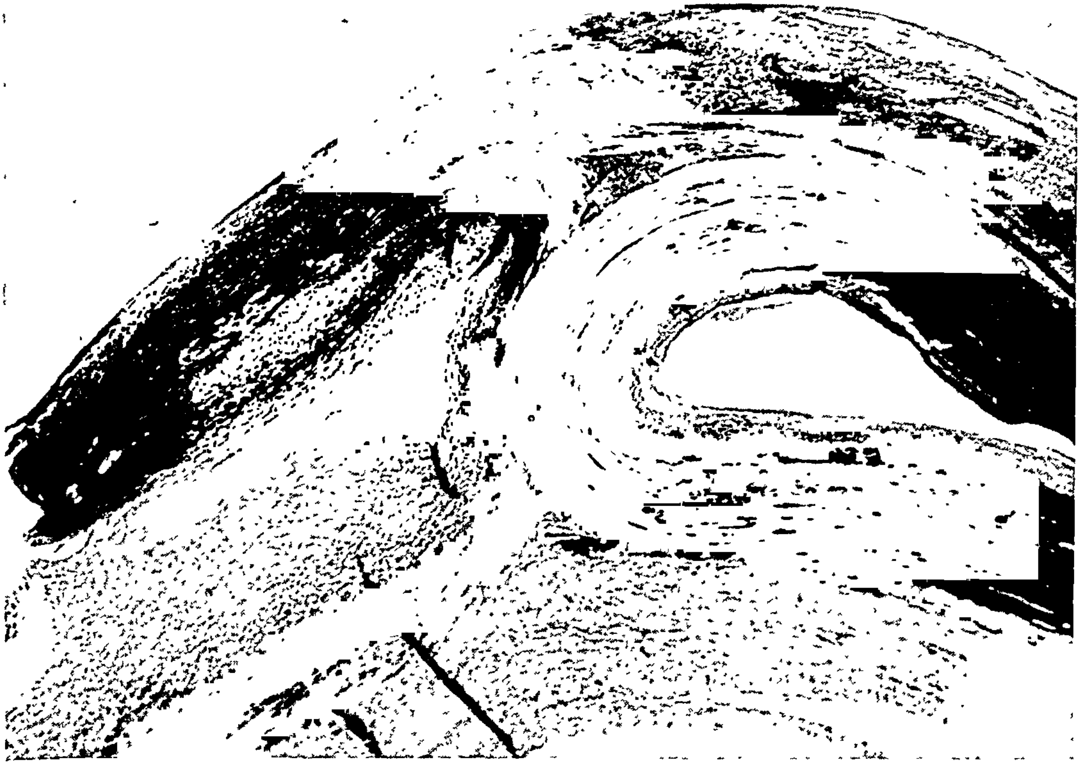


Fig. 5.—Case 2. Verhoeff's elastic tissue stain showing new elastic tissue formation in the secondary lumen toward the left side of the illustration. The new intimal connective ı this channel can be seen. (×20.)

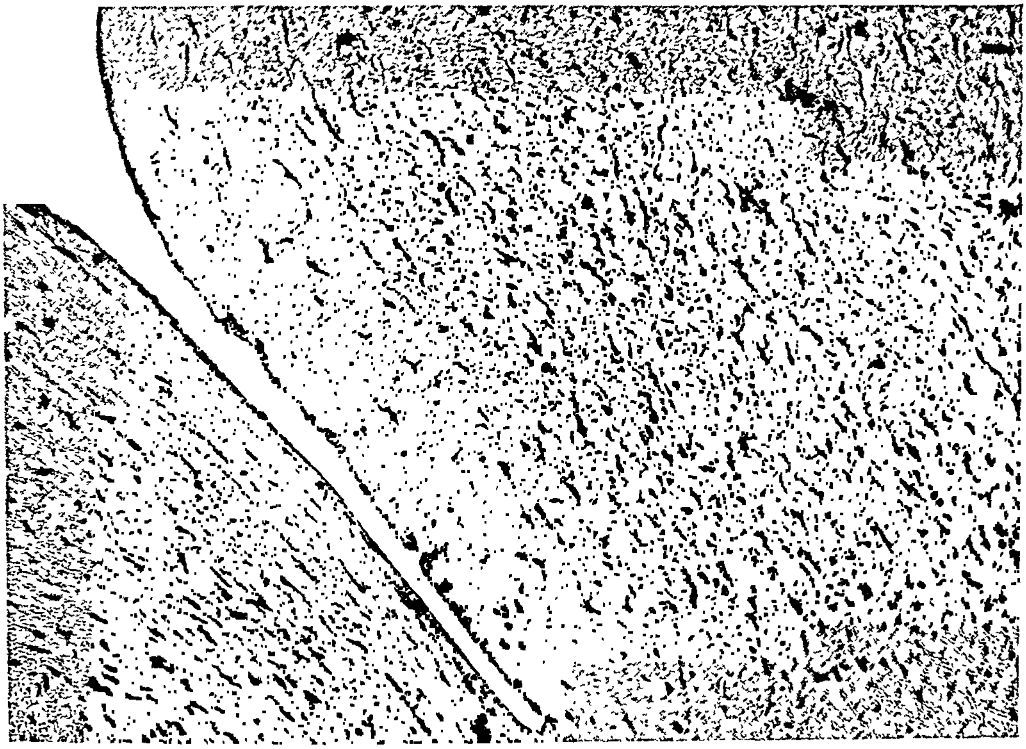


Fig. 6.—Case 2. Higher-power magnification of the newly formed intimal and subintimal connective tissue within the secondary channel. (×65.)

had become worse but there was no history of recent chest pain. The blood pressure at this time was 180/130 mm. Hg. Inspiratory rales were heard throughout both lungs and the heart sounds were faint. The record states that the patient's heart began to fibrillate, the blood pressure dropped to 80/56 mm. Hg and death occurred on Feb. 3, 1955.

At autopsy the significant findings included pulmonary edema, bilateral hydrothorax, bronchopneumonia, a benign duodenal ulcer with pancreatic penetration, left ventricular hypertrophy (heart weight—515 Gm.), and arteriolar nephrosclerosis. The thoracic aorta was the site of a fusiform dilatation measuring 19 by 5 cm. and encompassing 180 degrees of the posterolateral aortic circumference. This aneurysm began 4.5 cm. distal to the left subclavian artery and terminated 8 cm. above the coeliac axis. Viewed from the intimal surface there was a complete separation of intimal continuity just below the left subclavian artery (Fig. 8). The narrow portion of this intimal tear measured 1 cm. in length, while the widest portion took the form of a transverse oval defect measuring 5 by 2 cm. Both of these defects were filled with clotted blood. The distal intimal tear was circular in shape, had ragged margins, was filled with clotted blood, and measured 3 cm. in diameter. A triangular shaped pearly white intimal surface similar to that described in Case 1 extended for a distance of 4 cm. below this distal defect. Atherosclerosis was not present on this intimal surface. A separate intramural hematoma, measuring 1.5 by 2.5 by 2.5 cm. was noted at the level of the coeliac axis. This area showed no evidence of intimal rupture but did encroach upon the coeliac axis, right renal, and superior mesenteric arteries.

Microscopic examination of the aortic arch proximal to the tear revealed a thickened adventitia, intimal atherosclerosis, and no evidence of cystic medionecrosis. Sections taken from the area of the proximal tear showed marked intimal reparative activity (Fig. 9). The fibroblasts were characterized by pyknotic nuclei and showed active invasion of the clot. Verhoeff's elastic tissue stain revealed very scant amounts of elastic tissue within the newly formed, thickened intimal tissue. This intimal proliferation was histologically identical with experimental regeneration of the intima of the rabbit aorta as described by Prior and Hutter,[9] occurring about thirty-five days after intimal injury. The intramural clot was composed of acellular, amorphous, acidophilic debris, and organization of this material was proceeding very slowly from the outer coat of the aorta. The intima about the distal tear showed the same pattern of organization as described above in association with the proximal defect. The pearly white appearance of the triangular shaped intimal segment below the distal tear was caused by severe underlying cystic medionecrosis. The distal separate intramural hematoma appeared to be of more recent origin than the larger clot but showed intimal and adventitial connective tissue invasion into the hematoma.

CASE 4.—L. L. (A48 199), a 43-year-old white male farmer, was in good health until June 29, 1948, when he suffered the sudden onset of severe substernal pain during effort. He was immediately hospitalized and at that time the blood pressure was recorded as 230/170 mm. Hg in both arms. The temperature, pulse, and respirations were within normal limits. The pain persisted for four days and was characterized by infrascapular radiation. Laboratory data at this time revealed a total white blood cell count of 30,000 per c.mm., with 90 per cent polymorphonuclear leukocytes and 10 per cent lymphocytes. The red cell count was 8 million per cubic millimeter with 19 Gm. of hemoglobin. An electrocardiogram was stated to show left axis deviation. One week after hospitalization the blood pressure in both arms was 270/120 mm. Hg and the level in the legs was slightly lower. On July 8, 1948, eleven days after the acute episode, the blood pressure was not obtainable in the legs and they were cold and mottled blue-red in color. No arterial pulsations were present. A phlebotomy of 500 c.c. was carried out, following which warmth gradually returned to the legs, and the blood pressure was then recorded as 160/140 mm. Hg in both lower extremities.

The patient was transferred to Syracuse University Hospital on July 16, 1948, for further study. The patient was semiconscious and exhibited Cheyne-Stokes [illegible] The blood pressure was 235/130 mm. Hg in both legs. Pertinent physical fin [illegible] widening and elongation of the aortic shadow. No localized aneurysm was noted. The patient was fluoroscoped also and the final impression was that the aorta was arteriosclerotic, but because of the rapid increase in size, the possibility of dissection was suggested.

On Feb. 1, 1955, approximately seven and one-half months after onset of symptoms, the patient entered Syracuse General Hospital. During the previous month his dyspnea

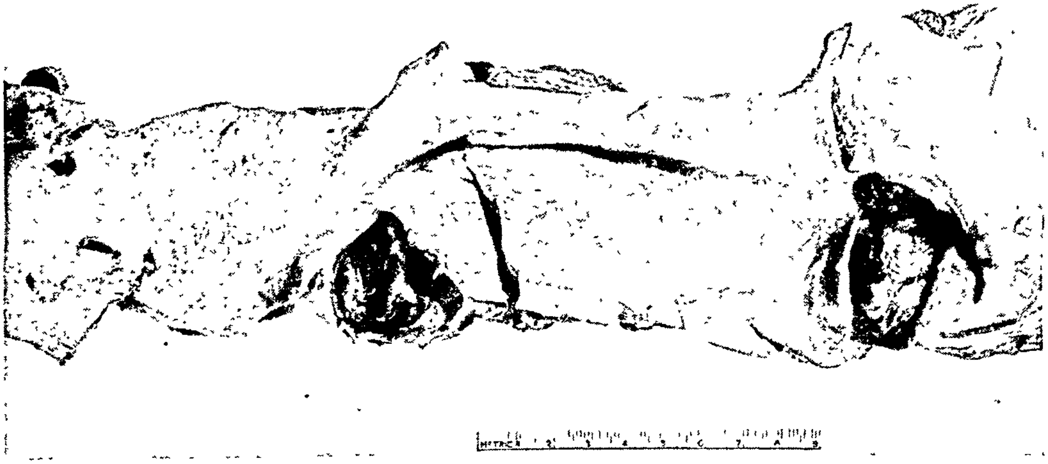


Fig. 8.—Case 3. Intimal aortic surface showing the two clot-filled intimal defects. The transverse laceration to the right of the distal defect is an artefact.

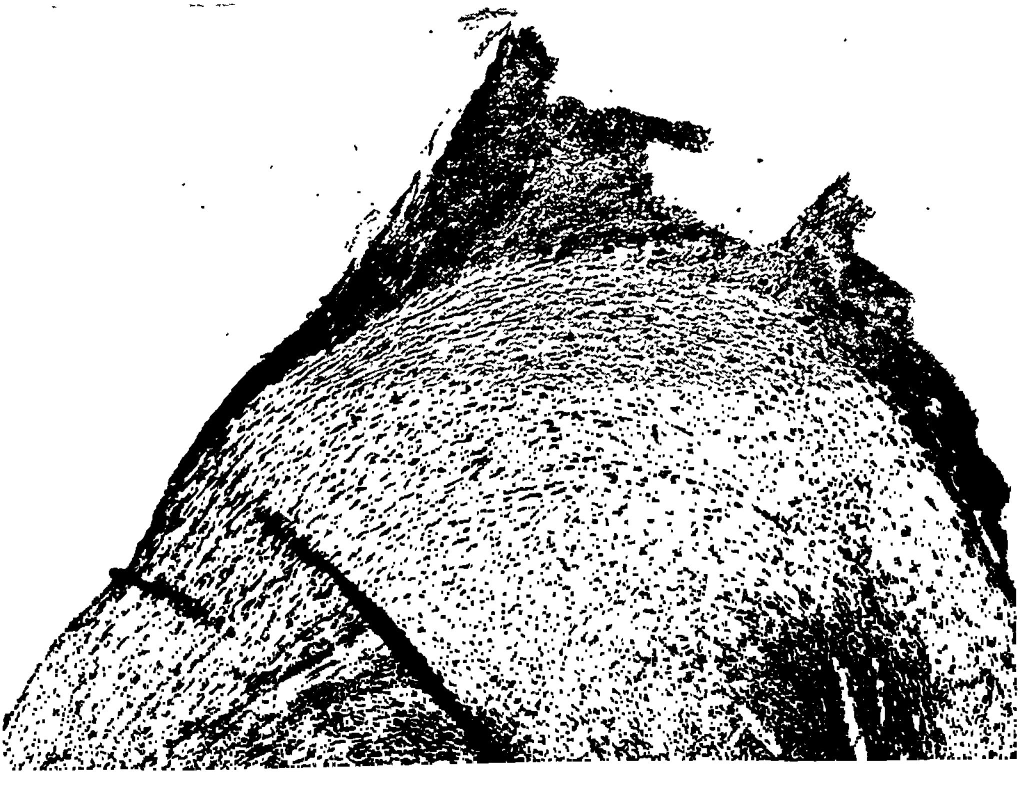


Fig. 9.—Case 3. Active intimal fibroblastic growth occurring about the proximal defect and extending into the blood clot. (×95.)

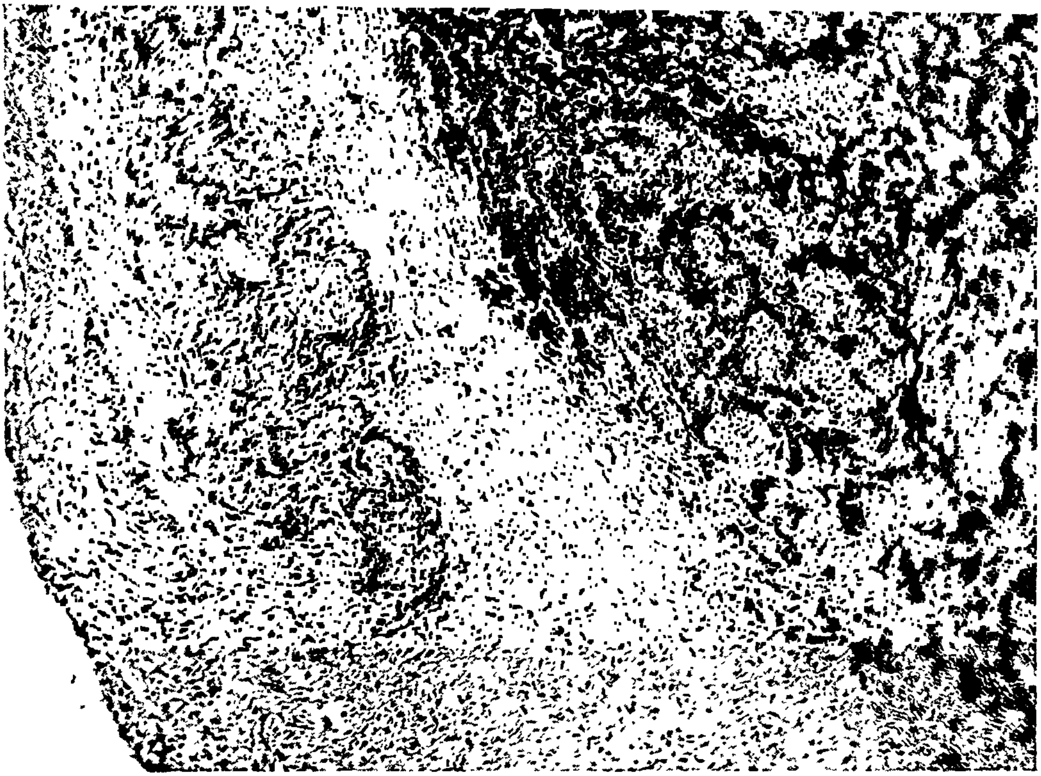


Fig. 10.—Case 4. Intimal and subintimal "granulation" type tissue with abundant new capillaries. The ruptured medial layer is noted in the left portion of the photomicrograph while the blood clot occupies the upper right half of the illustration. (×65.)

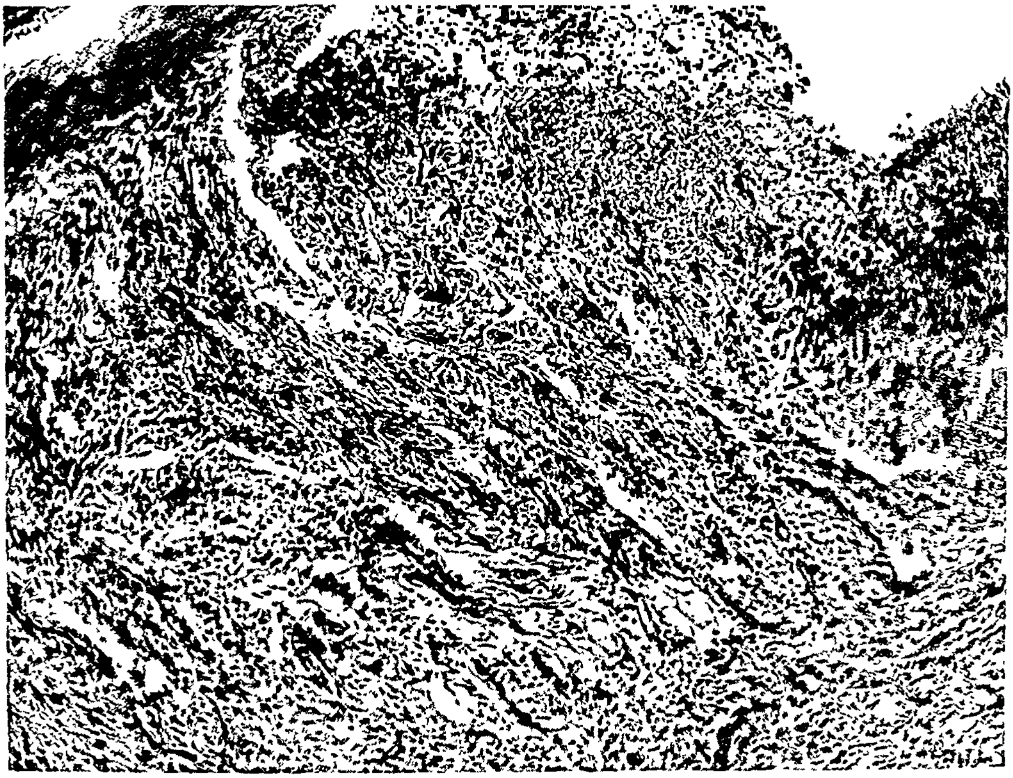


Fig. 11.—Case 4. Active granulation tissue invading the hematoma from the adventitial surface. (×70.)

widening and elongation of the aortic shadow. No localized aneurysm was noted. The patient was fluoroscoped also and the final impression was that the aorta was arteriosclerotic, but because of the rapid increase in size, the possibility of dissection was suggested.

On Feb. 1, 1955, approximately seven and one-half months after onset of symptoms, the patient entered Syracuse General Hospital. During the previous month his dyspnea

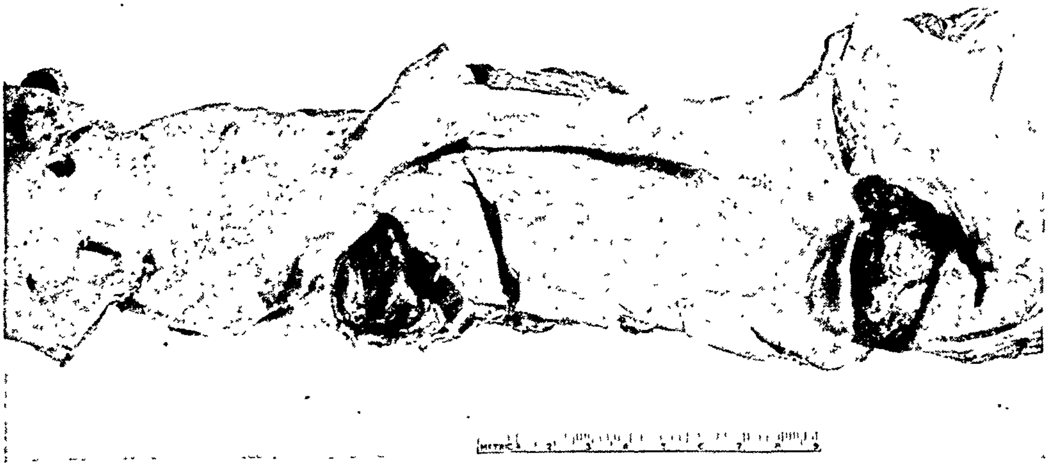


Fig. 8.—Case 3. Intimal aortic surface showing the two clot-filled intimal defects. The transverse laceration to the right of the distal defect is an artefact.

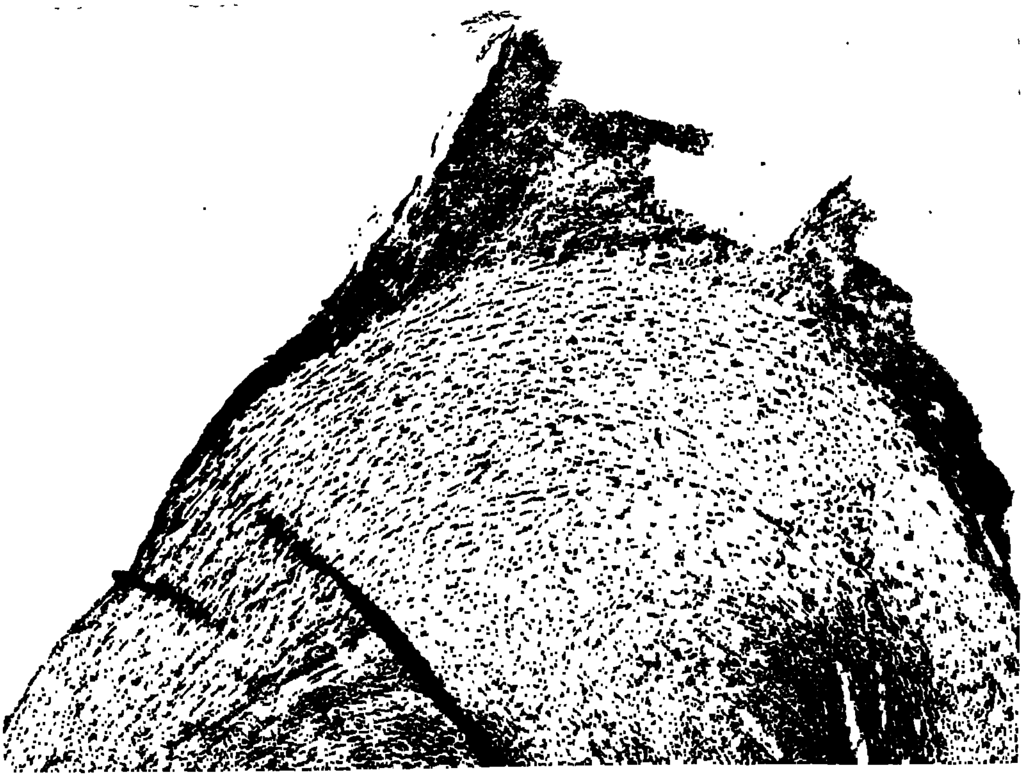


Fig. 9.—Case 3. Active intimal fibroblastic growth occurring about the proximal defect and extending into the blood clot. (×95.)

were minimal arteriovenous nicking, old white exudate in the right fundus, cardiac enlargement, and a systolic murmur along either side of the upper thoracic spine. The red blood and white blood cell counts were unchanged from the previous examination. An electrocardiogram disclosed left axis deviation, horizontal position of the heart, and digitalis effect. Roentgenogram of the chest showed left ventricular enlargement, enlargement of the arch of the aorta with increased tortuosity. The colloidal gold test on the spinal fluid was negative. During his hospital stay the patient received aminophylline, penicillin, paraldehyde, and a low sodium diet. On this regime the blood pressure in the arms dropped to 180/110 mm. Hg while the blood pressure in the legs remained at about 170/130 mm. Hg. The patient was discharged on July 30, 1948, with a tentative diagnosis of malignant hypertension, possible dissecting aortic aneurysm, and possible periarteritis nodosa. The patient died suddenly a few hours following discharge.

At autopsy, the significant findings included a left hemothorax, atelectasis of the left lung, patchy areas of myocardial fibrosis, chronic passive congestion of the liver and spleen, and arteriolar nephrosclerosis. The heart showed left ventricular hypertrophy, the left ventricle measuring 2 cm., and the right measuring 0.6 cm. in thickness. Inspection of the interior of the aorta disclosed an intimal tear located just distal to the origin of the left subclavian artery. This tear led into an intramural aortic dissection which extended posteroinferiorly to end at a point just below the origin of the renal arteries. This secondary channel was filled with clotted blood which exerted a narrowing effect upon the true aortic lumen. No point of re-entry into the true lumen was found. The renal arteries, left subclavian, left common carotid, celiac axis, and mesenteric arteries were not compromised by this dissection. There was an irregular tear on the adventitial surface of the aneurysm which measured 3 cm. in diameter and which was the point at which the dissection had ruptured into the left hemothorax—there being about 3,000 c.c. of blood in this space.

Microscopic examination of the sections from the aorta showed severe cystic medionecrosis at all levels and moderately severe atherosclerosis throughout. The dissection had occurred at the junction of the middle and outer thirds of the medial coat. The mural hematoma presented an amorphous eosinophilic appearance due to the total autolysis of the red blood cells. Reparative activity was marked about the site of the intimal rupture (Fig. 10). The fibroblasts in this area were swollen, contained pale vesicular nuclei, and showed no tendency toward an orderly orientation. It is of interest that this newly forming intimal and subintimal tissue contained many new capillaries, a most unusual observation in our experience. Furthermore, it has been demonstrated that the experimentally induced traumatic types of lesion within the rabbit aorta show no new capillaries during the phase of intimal and subintimal regeneration.[9] Organization of the intramural hematoma from the outer surface of the vessel was, however, characterized by abundant amounts of typical granulation tissue (Fig. 11). In addition to the newly formed fibroblasts and blood capillaries, there were large numbers of macrophages containing phagocytized iron and rare multinucleated giant cells.

CASE 5.—V. L. (A53-229), an 86-year-old white woman, entered Syracuse General Hospital on Nov. 21, 1947, with a chief complaint of the sudden onset of severe left chest pain that penetrated to the back and radiated to the left shoulder. Physical examination disclosed the blood pressure to be 200/110 mm. Hg and there was a weak, fast, irregular pulse. There were dullness and absent breath sounds over the base of the left lung with hyperresonance in the remainder of the lung. A few moist basilar rales were noted in the right lung. The heart was enlarged and mitral and aortic murmurs were described. The abdominal examination was negative. Roentgenograms of the chest were interpreted as being consistent with a pulmonary infarction in the left lower lobe. The patient was discharged improved.

She re-entered Syracuse General Hospital on Oct. 25, 1953, with complaints primarily related to the gastrointestinal system. Belching, flatulence, and dysphagia of several years' duration were present. No history of weight loss, diarrhea, or melena was obtained. The blood pressure at this time was 220/110 mm. Hg. Moist basilar rales and dullness were minimal arteriovenous nicking, old white exudate in the right fundus, cardiac enlargement, and a systolic murmur along either side of the upper thoracic spine. The red blood and white blood cell counts were unchanged from the previous examination. An electrocardiogram disclosed left axis deviation, horizontal position of the heart, and digitalis effect. Roentgenogram of the chest showed left ventricular enlargement, enlargement of the arch of the aorta with increased tortuosity. The colloidal gold test on the spinal fluid was negative. During his hospital stay the patient received aminophylline, penicillin, paraldehyde, and a low sodium diet. On this regime the blood pressure in the arms dropped to 180/110 mm Hg while the blood pressure in the legs remained at about 170/130 mm. Hg. The patient was discharged on July 30, 1948, with a tentative diagnosis of malignant hypertension, possible dissecting aortic aneurysm, and possible periarteritis nodosa The patient died suddenly a few hours following discharge.

At autopsy, the significant findings included a left hemothorax, atelectasis of the left lung, patchy areas of myocardial fibrosis, chronic passive congestion of the liver and spleen, and arteriolar nephrosclerosis The heart showed left ventricular hypertrophy, the left ventricle measuring 2 cm, and the right measuring 0.6 cm. in thickness. Inspection of the interior of the aorta disclosed an intimal tear located just distal to the origin of the left subclavian artery. This tear led into an intramural aortic dissection which extended posteroinferiorly to end at a point just below the origin of the renal arteries This secondary channel was filled with clotted blood which exerted a narrowing effect upon the true aortic lumen. No point of re-entry into the true lumen was found. The renal arteries, left subclavian, left common carotid, celiac axis, and mesenteric arteries were not compromised by this dissection There was an irregular tear on the adventitial surface of the aneurysm which measured 1.3 cm in diameter and which was the point at which the dissection had ruptured into the left hemothorax—there being about 3,000 c.c. of blood in this space

Microscopic examination of the sections from the aorta showed severe cystic medionecrosis at all levels and moderately severe atherosclerosis throughout The dissection had occurred at the junction of the middle and outer thirds of the medial coat The mural hematoma presented an amorphous eosinophilic appearance due to the total autolysis of the red blood cells Reparative activity was marked about the site of the intimal rupture (Fig. 10). The fibroblasts in this area were swollen, contained pale vesicular nuclei, and showed no tendency toward an orderly orientation. It is of interest that this newly forming intimal and subintimal tissue contained many new capillaries, a most unusual observation in our experience. Furthermore, it has been demonstrated that the experimentally induced traumatic types of lesion within the rabbit aorta show no new capillaries during the phase of intimal and subintimal regeneration.[9] Organization of the intramural hematoma from the outer surface of the vessel was, however, characterized by abundant amounts of typical granulation tissue (Fig. 11). In addition to the newly formed fibroblasts and blood capillaries, there were large numbers of macrophages containing phagocytized iron and rare multinucleated giant cells.

CASE 5.—V. E. (A53 229), an 86 year old white woman, entered Syracuse General Hospital on Nov. 21, 1947, with a chief complaint of the sudden onset of severe left chest pain that penetrated to the back and radiated to the left shoulder. Physical examination disclosed the blood pressure to be 200/110 mm Hg and there was a weak, fast, irregular pulse. There were dullness and absent breath sounds over the base of the left lung with hyperresonance in the remainder of the lung. A few moist basilar râles were noted in the right lung. The heart was enlarged and mitral and aortic murmurs were described. The abdominal examination was negative. Roentgenograms of the chest were interpreted as being consistent with a pulmonary infarction in the left lower lobe. The patient was discharged improved

She re entered Syracuse General Hospital on Oct. 25, 1953, with complaints primarily related to the gastrointestinal system. Belching, flatulence, and dysphagia of several years' duration were present. No history of weight loss, diarrhea, or melena was obtained. The blood pressure at this time was 220/110 mm. Hg. Moist basilar râles and dullness

Microscopic study of the aorta disclosed severe atherosclerosis along its entire length and considerable medial calcification was also present Cystic medionecrosis was never prominent, only rare minute foci being observed The major portion of the blood clot within both secondary channels had been converted into a hyalinized connective tissue with considerable calcification. Both of the secondary lumens were lined by dense avascular connective tissue and well defined endothelial cells (Fig 12). With the use of Verhoeff's technique, very few elastic fibrils were demonstrable It was apparent that the cleavage site in these *old* dissections had occurred roughly at the junction of the outer and middle thirds of the media The vasa vasorum showed no change judged to be of significance

### DISCUSSION

A review of the literature suggests that most of the clinical and pathologic information concerning healed dissecting aneurysms is to be found in studies of reports of isolated cases[6, 7, 10, 19] Very few reviews of a large series of cases are available. The most comprehensive study of the chronic-type cases was carried out by Shennan[20] who collected 79 examples of healed aortic aneurysm from the literature. Survival for periods longer than one year was recorded in 16 of these cases Shennan points out that re-entry of the dissection into the original lumen is a prerequisite for survival When it did not occur, 96 per cent of the patients died within five weeks, whereas, when circulation was re-established in this way only 28 per cent died within the first five weeks Shennan's statistics also show that if the patient survives for more than five weeks, re-entry has taken place in 90 per cent (66 of 73 cases) He pointed out that re-entry into the original lumen is particularly liable to occur when the dissection reaches some obstruction, due to increased pressure within the aneurysmal sac The most common cause of death, in Shennan's series of chronic dissecting aneurysms, was heart failure, followed next frequently by hemorrhage from the aneurysm and cerebral hemorrhage While the pathogenesis of the cardiac failure is not always clear, it has occurred to us that cardiac failure may often come about in a manner comparable to that associated with coarctation of the aorta, the intramural hemorrhage producing extreme narrowing of the original aortic lumen Case 3 in our series may illustrate this type of failure since it was possible to get only the tip of the little finger into the original aortic lumen The heart weighed 515 Gm and the terminal blood pressure in this patient was 180/130 mm Hg Left ventricular hypertrophy was recorded in 65 out of 79 patients of Shennan's with healed aneurysms and he ascribed this as due either to increased work placed on the left ventricle by the new channel or due to compression of the original lumen by the new sac Levinson and his colleagues,[16] in their study of 58 cases of dissecting aneurysm, recorded 15 patients who survived periods of from three months to eight years They point out that this large number of cases in the chronic group is a more encouraging finding than Weiss[21] had noted in an earlier study Weiss stated that healing occurred in about 10 per cent of all patients with dissecting aneurysm, chiefly by means of re-entry of the false passage into the true lumen at a lower level.

Considerable evidence exists to indicate that the ante-mortem diagn of dissecting aneurysm is being made with increased frequency.[22] While were present posteriorly over both lungs. Cardiac enlargement, aortic and mitral systolic murmurs were noted. The liver edge was stated to be 4 inches below the right costal margin in the midclavicular line. Weakness of the left leg and weak, thready pulses were present in both lower extremities. Laboratory data disclosed the hematocrit to be 21 per cent; the red blood cell count 2.2 million per cubic millimeter; white blood cell count 19,500 per c.mm., with 86 per cent polymorphonuclear leukocytes, and 14 per cent lymphocytes. Microcytosis was prominent in the smear. The urine examination was negative except for a 2+ albumin. The patient died on Oct. 26, 1953, after a steady downhill course.

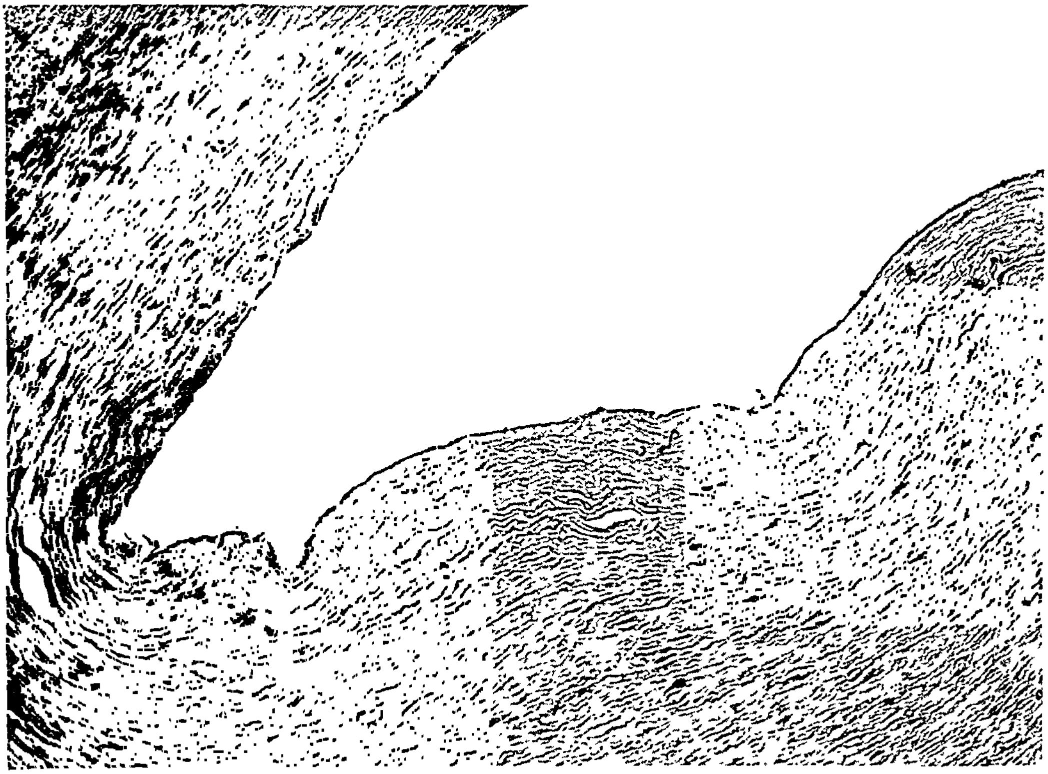


Fig. 12.—Case 5. Secondary channel showing the dense connective tissue lining. (×65.)

At autopsy the following findings were noted: adenocarcinoma of the stomach with metastasis to the liver, adenocarcinoma of the cecum arising in a sessile adenomatous polyp, pulmonary edema, and chronic pericarditis. The heart weighed 345 Gm. and showed no valvular abnormalities. Inspection of the interior of the aorta disclosed a transverse slit-like opening along the medial aspect of the midthoracic descending aorta. This opening led into a cavity within the aortic wall which was filled with cartilaginous-like material and extended 3 cm. superiorly and 3 cm. inferiorly. No other opening into this dissection was found. The greatest diameter of this aortic intramural mass was 1.5 cm. Inferiorly, just proximal to the diaphragm, a second intimal defect and intramural dissection was noted. This was also filled with cartilaginous-like material, was located on the medial wall, and measured 1.5 cm. in diameter and 7 cm. in length. The intimal slit-shaped defect was located approximately in the midportion of the dissection. Although these two intramural healed dissections were separated from each other by a distance of 1 to 2 cm., it is our impression that they were at one time contiguous and subsequently the channel became obliterated in much the same fashion as in Case 1 of the present series.

false passage below In instances in which the lesion was well localized at or below the left subclavian artery, they combined this procedure with excision of the segment involved in the origin of the dissection, followed by end-to-end anastomosis or insertion of an aortic homograft Two important points stressed by these authors were control of hypertension, both during and following surgery, and the necessity of early intervention in the acute cases

## SUMMARY

The clinical and pathologic characteristics of 5 fatal cases of chronic (healed) dissecting aneurysms have been described Survival time in these patients ranged from sixty-three days to several years, and the pathologic changes varied from imperfectly organized intramural hematomata to secondary endothelial lined channels (double barrel aortas) with abundant elastification and pronounced intimal and subintimal proliferative changes A survey of the diagnostic measures and the surgical principles which have been utilized in cases of dissecting aneurysms has been presented

## REFERENCES

1 Gurin, D, Bulmer, J W, and Derby, R Dissecting Aneurysm of the Aorta Diagnosis and Operative Relief of Acute Arterial Obstruction Due to This Cause, New York State J Med 35 1200 1202, 1935
2 Johns, T N P Dissecting Aneurysm of the Abdominal Aorta Report of Case With Repair of Perforation, Ann Surg 137 232 235, 1953
3 Shaw R S Acute Dissecting Aortic Aneurysm Treatment by Fenestration of the Internal Wall of the Aneurysm, New England J Med 253 331 333, 1955
4 DeBakey, M E, Cooley, D A, and Creech, O, Jr Surgical Considerations of Dissecting Aneurysm of the Aorta, Ann Surg 142 586 612, 1955
5 Weiss, S, Kinney, T D, and Maher, M M Dissecting Aneurysm of the Aorta With *Experimental Atherosclerosis*, *Am J M Sc* 200 192 202, 1940
6 Cassidy, M, and Pinniger, J Healed Dissecting Aneurysm, Brit Heart J 8 130 140, 1946
7 Blumenthal, J S Dissecting Aneurysm of the Aorta, Minnesota Med 33 255 258, 1950
8 Prior J T, and Jones, D B Structural Alterations Within the Aortic Intima in Infancy and Childhood, Am J Path 28 937 951, 1952
9 Prior, J T, and Hutter, R V P Intimal Repair of the Aorta of the Rabbit Following Experimental Trauma, Am J Path 31 107 123 1955
10 Gouley, B A and Anderson, E Chronic Dissecting Aneurysm of the Aorta Simulating Syphilitic Cardiovascular Disease, Notes on the Associated Aortic Murmurs, Ann Int Med 14 978 990, 1940
11 Hoskin, J, and Gardner, F Silent Dissection of the Aorta, Brit Heart J 8 141 146, 1946
12 Jones, A M, and Langley, F A Chronic Dissecting Aneurysms, Brit Heart J 8 191 199, 1946
13 Coston, A S Healed Dissecting Aneurysm, Arch Path 48 309 315, 1949
14 Spicknall, C G, and Binford, C H Healed Dissecting Aneurysm of the Aorta With Signs of Aortic Insufficiency, Mil Surgeon 102 47 49, 1948
15 Smith, J C and Sancetta, S M Healed Dissecting Aneurysm of the Aorta Erroneously Diagnosed Paramediastinal Effusion, Death Following Attempted Operation, Circulation 1 792 796, 1950
16 Levinson, D C, Edmeades, D T, and Griffith, G C Dissecting Aneurysm of the Aorta Its Clinical, Electrocardiographic, and Laboratory Findings, Circulation 1 360 378, 1950
17 Pomerantz, H Z, Darragh, J H, and Mallory, G K Recurring Dissecting Aneurysm of the Aorta, Canad M A J 67 339 342 1952
18 Hunter, W C, and Lium, J H Unusual Pathologic Manifestations of Dissecting Aortic Aneurysms, Am J Path 28 1035 1058, 1952
19 Halpert, B, and Brown, C A Dissecting Aneurysms of the Aorta, Arch Path 60 378 385, 1955

history and physical examination are of great importance, their value may often be restricted due to the condition of the patient. The story of excruciating epigastric or precordial pain associated with atypical patterns of radiation is strongly suggestive of dissection. Clinical evidence of shock in the presence of sustained hypertension is usually present. Paresthesias and alterations in the circulation of the extremities are not uncommon. According to Levinson[16] there is no specific electrocardiographic pattern in dissecting aneurysm, absence of electrocardiographic changes of myocardial infarction tending to support the diagnosis of aortic dissection in equivocal cases. The most useful diagnostic aid appears to be roentgenographic examination of the thorax. Intimal calcification makes measurement of the thickness of the aortic wall possible. Lodwick[23] states that the wall normally measures 2 to 3 mm. in thickness and that, while increases up to 1 cm. are suggestive, widening over 1 cm. is diagnostic of an intramural aortic hematoma. In the absence of intimal calcification, it is impossible to measure directly the thickness of the wall and the diagnosis becomes much more difficult. A widened aortic shadow may be due to elongation and stretching associated with arteriosclerosis alone, and a left-anterior oblique or right-posterior oblique view demonstrates this best according to Levene and co-workers.[24] Diffusely fusiform syphilitic aneurysms may also be confused with dissecting aneurysms on a roentgenologic basis. The margin of this type of aneurysm forms a gentle curve while in dissecting aneurysm, although the involved portion of the aorta is widened, it is of such a nature as to produce a straightening of the aortic curve in the involved segment. This straightened wall shows numerous irregularities, as in Case 3, due to larger collections of intramural blood clots at these sites (Fig. 7). The widened segment swings back to the mediastinum at an angle, and not in a curve as with the syphilitic aneurysm.

There is considerable evidence of renewed interest in the subject of dissecting aneurysm in view of the great progress of cardiovascular surgery in recent years. Surgery has been carried out in both the acute and chronic varieties of dissecting aneurysm and there is reason to believe it may have a very real role in the treatment of a condition previously considered to be hopeless. The single cases of surgical intervention reported by Gurin and collaborators,[1] Johns,[2] and by Shaw,[3] were all followed by death from renal insufficiency within the first ten days after surgery. Recently, however, DeBakey and his colleagues[4] have reported a series of 6 patients who were surgically treated, with only 2 fatalities. The duration of the dissection in this series of cases ranged from two days to thirteen months. The rationale of the surgical procedures proposed by DeBakey and his co-workers is based upon the principle that re-entry of the dissected passage at some distal point in the aorta permits restoration of peripheral circulation and removes the increasing tension upon the outer wall which predisposes to rupture. Several surgical approaches utilizing similar principles were employed. In those cases in which the dissection began in the ascending or descending arch, a re-entry passage was created into the aortic lumen with obliteration of the

# PNEUMOPERITONEUM AS A SPACE-OCCUPYING PROCEDURE IN CONJUNCTION WITH PULMONARY RESECTION

HOWARD A. BUECHNER, M.D., MORTON M. ZISKIND, M.D.,
AND LAWRENCE H. STRUG, M.D.
NEW ORLEANS, LA.

THE place of lobectomy and segmental resection in the treatment of pulmonary tuberculosis is now clearly established.[1] Techniques of these procedures have been well delineated, but the problem of dealing with the residual space resulting from removal of pulmonary tissue has not been satisfactorily solved. The need for a space-filling procedure to precede, accompany, or follow pulmonary resection has concerned itself chiefly with three issues; namely, (1) conservation of pulmonary function, (2) elimination of dead space and ensuing complications resulting from failure of remaining lobes to overdistend and completely fill the hemithorax, and (3) prevention of reactivation of residual tuberculous foci.

The original idea that simple pulmonary overdistention resulted in a loss of function comparable to that produced by diffuse obstructive emphysema has now been largely discarded. On the basis of physiologic studies by Gaensler and Strieder,[2] and others,[3, 4] it has been established that postresection overdistention actually costs little in terms of impaired function of the remaining pulmonary tissue. From this standpoint alone, therefore, a space-occupying procedure hardly appears to be necessary. In fact, the commonly employed postresection thoracoplasty will usually decrease, rather than preserve pulmonary function,[2, 3] by partially collapsing the remaining lung tissue and impairing costal motion.

On the basis of these findings, a steady trend away from the use of space-occupying procedures has developed, with their selected employment only when full inflation of the remaining lung fails to occur and obliteration of dead space becomes a necessity. Chamberlain and co-workers[5] have stressed the importance of rapid elimination of all residual pleural space as a basic surgical principle if infection is to be avoided, and have stated[6] that "obliteration of the pleural dead space by complete and immediate re-expansion is the most important single factor in the postoperative management." They have indicated[6] that delayed or incomplete re-expansion will be encountered in about 6 per cent of the patients undergoing segmental resection, while Goldman and his colleagues[7] have seen this complication in 17 per cent of their patients.

---

From the Tuberculosis Service and the Thoracic Surgery Service, Veterans Administration Hospital, New Orleans, La., and the Department of Medicine, Tulane University of Louisiana

Received for publication Jan 3, 1956.

20. Shennan, T.: Dissecting Aneurysms, Medical Research Council, Special Report Series, No. 193, His Majesty's Stationary Office, London, 1934.
21. Weiss, S.: The Clinical Course of Spontaneous Dissecting Aneurysm of the Aorta, M. Clin. North America 18: 1117-1141, 1935.
22. Bauersfield, S. R.: Dissecting Aneurysm of the Aorta: a Presentation of 15 Cases and a Review of the Recent Literature, Ann. Int. Med. 26: 873-889, 1947.
23. Lodwick, G. S.: Dissecting Aneurysms of the Thoracic and Abdominal Aorta. Report of 6 Cases With a Discussion of Roentgenologic Findings and Pathologic Changes, Am. J. Roentgenol. 69: 907-925, 1953.
24. Levene, G., Burke, E. N., and Arnois, D. C.: Roentgenologic Diagnosis of Aneurysms of the Thoracic Aorta With Particular Reference to Study of the Right Posterior Oblique Position, Am. J. Roentgenol. 72: 1004-1016, 1954.

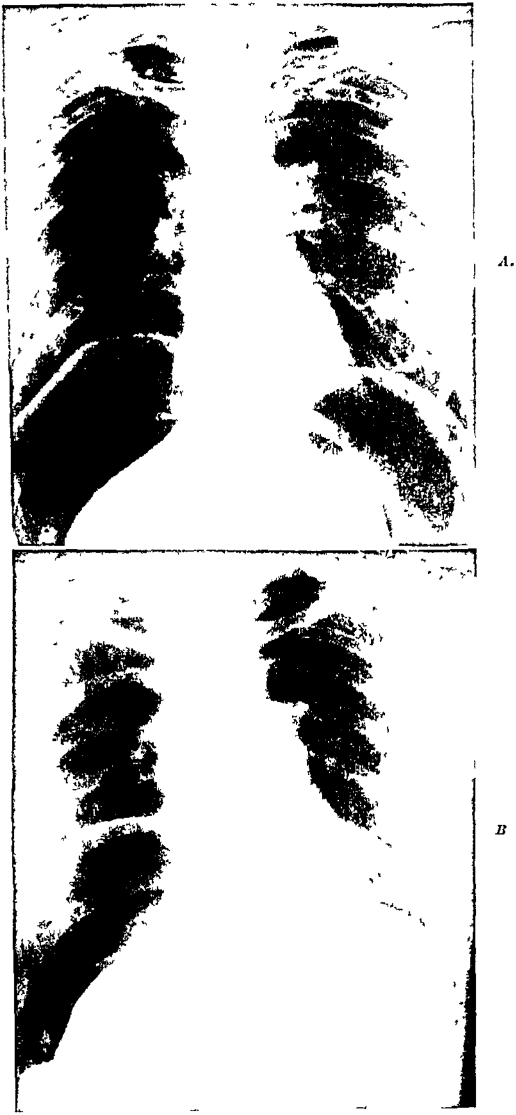


Fig 1—*A*, Chest film taken four days prior to right upper lobectomy, showing moderate degree of pneumoperitoneum *B*, Chest film taken on fourth postoperative day. Note complete redistribution of air beneath the right diaphragm producing selective elevation.

If a lobectomy or a lobectomy and resection of a superior segment is carried out, the remaining pulmonary tissue will fail to fill completely the hemithorax in an even greater number of cases.

When dead space persists, it is usually obliterated by employment of a modified thoracoplasty or some type of plombage procedure, thus requiring a second major operation. It seems logical that the incidence of residual postresection dead space might be decreased by a procedure which automatically reduces the total volume of the hemithorax before and during surgery and thus diminishes the space which the remaining pulmonary tissue must fill. If this can be accomplished without resort to multiple operations, it would appear even more desirable. As will be pointed out in subsequent discussion, artificial pneumoperitoneum seems to serve this purpose well.

The question as to whether or not there is a significant risk of reactivation of residual disease in portions of lung which are subjected to sudden stretching, as the remaining pulmonary tissue is required to overdistend in an attempt to fill the chest, is still a controversial matter. Under the protection of specific tuberculostatic drugs the incidence of reactivated tuberculosis does not appear to be influenced by postresection hyperinflation,[3] and yet overdistention is the exact opposite of the relaxation of lung tissue produced by various collapse procedures long accepted as beneficial in promoting healing of tuberculous lesions. In any event, until further information is at hand, it would appear wise not to brush this question aside and place sole dependence on antimicrobial therapy to prevent the disaster of reactivated disease when an additional measure is available that will reduce the risk to a minimum.

The readiness with which space-filling procedures have fallen into disuse, in spite of the risks involved, is indicative that those which have been usually employed are each associated with significant disadvantages. "Partial" or "tailoring" thoracoplasty has the obvious disadvantages of producing deformity, extending the duration of the operative procedure, or requiring a second operation and reducing pulmonary function for reasons already stated.

Phrenic crush decreases pulmonary function by producing diaphragmatic paralysis and thus contributes to the development of postoperative complications.

Various plombage procedures avoid the production of thoracic deformity and probably do not cause significant further reduction of function, but require additional major surgery and are associated with important complications including fluid formation, hemorrhage, and infection.

The value of pneumoperitoneum as a space-occupying procedure in conjunction with pulmonary resection appears to have been largely overlooked. Although, Bickford and co-workers,[8] in 1951, advocated the employment of pneumoperitoneum in this way, they combined it with phrenic crush, thus adding the disadvantages of reduced pulmonary function and increased susceptibility to postoperative atelectasis. Perhaps for this reason the procedure has never attained widespread usage. Our attention was first focused on this subject by observing patients in whom pneumoperitoneum had been established as a therapeutic measure and resection carried out later. We were intrigued

A total of twelve cases of lobectomy or lobectomy plus segmental resection has been managed in this fashion, and one of us (L H S ) has had additional experience with the procedure at another hospital In this series, pneumoperitoneum has given excellent results In only 1 case did the remaining lung fail to fill the hemithorax promptly and completely This patient developed a rather intense postoperative pleural reaction and a small apical air space persisted for slightly more than two weeks before complete re-expansion was attained No complications attributable to pneumoperitoneum have been seen, nor has there been any evidence of reactivation of residual disease Significant complications were encountered in only 1 patient who developed postoperative atelectasis followed by a bronchopleural fistula and empyema Another patient exhibited transient segmental atelectasis which resolved spontaneously The manner in which the intraperitoneal air redistributes itself following surgery and produces the exact desired result is clearly illustrated in the accompanying illustrations (Figs 1 and 2)

In an attempt to set up a comparison for the group just discussed, the last 12 patients having lobectomy or segmental resection for pulmonary tuberculosis performed at this hospital, in which neither accompanying thoracoplasty nor concomitant pneumoperitoneum was employed, were reviewed Five of these patients had delayed re-expansion of the remaining pulmonary tissue, which in two instances required reinsertion of chest tubes and reapplication of suction In an additional patient, re-expansion was permanently incomplete and thoracoplasty was required One patient developed early reactivation of tuberculosis Three patients developed significant postoperative atelectasis, and four bronchopleural fistulas were seen Only 3 of the 12 patients were completely free of complications, although a satisfactory result was eventually attained in all cases It seems noteworthy that 1 of the patients just mentioned, with previously induced therapeutic pneumoperitoneum, had this procedure deliberately discontinued at the time of surgery by withdrawal of air from the abdominal cavity Interestingly enough, this patient developed atelectasis and incomplete re-expansion of the remaining pulmonary tissue

While these two series are admittedly small, the striking difference in results appears to be significant and certainly justifies continued use of pneumoperitoneum in this fashion

An alternate method of employing pneumoperitoneum as a space occupying procedure on a more selective basis might be more appealing to some physicians If, at the time of surgery, operative findings seem to indicate that the remaining lung tissue will not readily overdistend and completely fill the hemithorax, a pneumoperitoneum can be induced immediately or during the early postoperative period, thus utilizing the procedure only in selected patients Pneumoperitoneum can be induced at the time of surgery by introducing a needle through the diaphragm from within the open chest cavity This method, however, is mentioned only to condemn it The danger of injury to the liver and air embolism appears to be so great that we feel this technique should never be used A somewhat safer method involves a stab wound of by the manner in which the air distributed itself selectively beneath the diaphragm on the operative side and reduced the size of the hemithorax. Pneumoperitoneum was thus found to function very efficiently as a space-filling procedure and presented obvious advantages. It produces no deformity, does not require additional surgery, is not associated with significant complications, causes no important reduction of pulmonary function, usually produces selective elevation of the diaphragm on the operative side, is a completely reversible procedure, and allows gradual subsequent relaxation of the remaining lung.

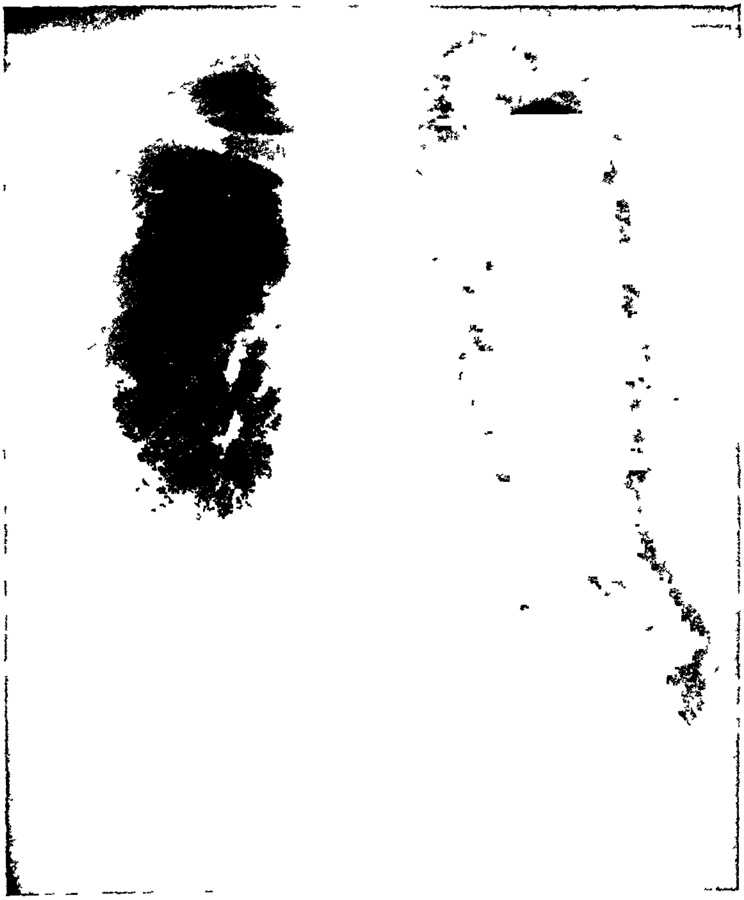


Fig. 1C.—Chest film taken eleven months after lobectomy. The right diaphragm remains elevated but functions well. True overdistention of the right lung has never occurred.

Recently, it has been our practice to employ pneumoperitoneum in conjunction with some types of pulmonary resection as a routine procedure. When it has been decided that a patient is to undergo a lobectomy or resection of multiple segments, pneumoperitoneum is induced (usually about one month prior to surgery) and a moderate elevation of the diaphragms is produced during the course of four to six refills. Following surgery, pneumoperitoneum is maintained for about two months, the first refill being given ten to fourteen days following the operation and weekly thereafter.

the diaphragm with the insertion of a small catheter, introduction of air beneath the diaphragm, and subsequent closure of the diaphragmatic opening by means of a purse-string suture. In spite of the advantage of selectivity offered by these methods, we do not favor introduction of pneumoperitoneum at the time of surgery or during the immediate postoperative period. We prefer the procedure now in use since it allows proper evaluation of the patient's tolerance of pneumoperitoneum and the benefits to be expected before surgery is carried out.

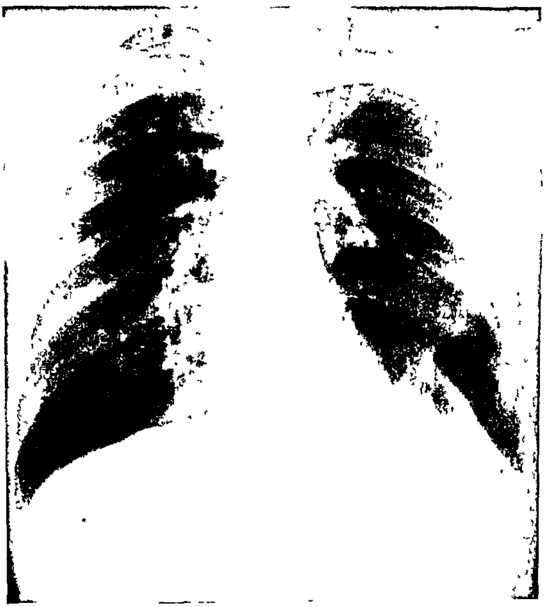


Fig. 2C.—Chest film taken eleven months after lobectomy. A pleurodiaphragmatic adhesion remains but the diaphragm functions well.

Some of our associates have objected to the use of pneumoperitoneum, in this manner, with claims that the presence of air in the peritoneal cavity and associated elevation of the diaphragms renders surgery technically more difficult and contributes to prolonged and irregular anesthesia. We can only state that our experiences have failed thus far to confirm these objections in a single instance.

## SUMMARY

1. The value of lobectomy and segmental resection in the treatment of tuberculosis has been clearly established, but the problem of dealing with the residual space resulting from removal of pulmonary tissue continues to tax the ingenuity of the thoracic surgeon.

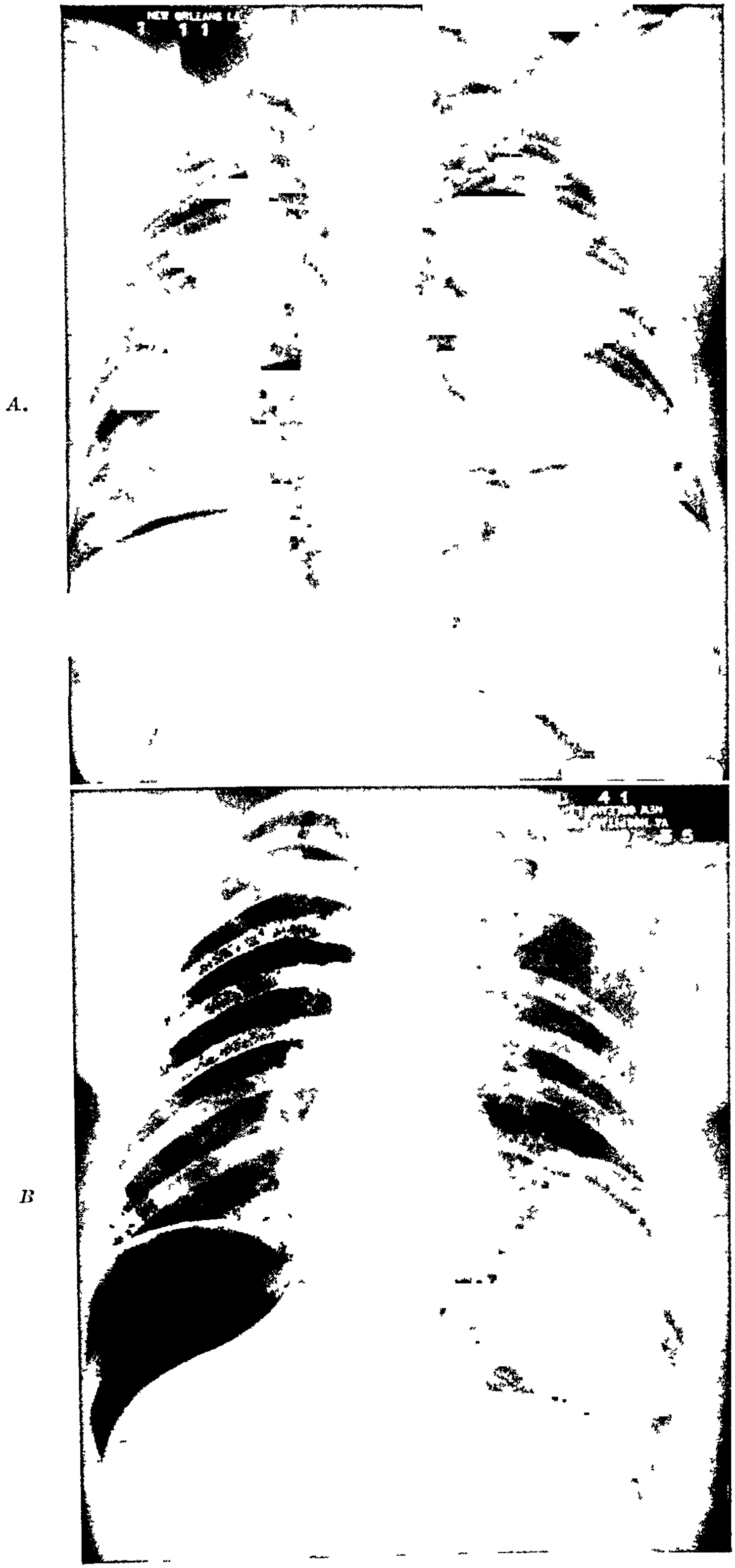


Fig. 2.—*A*, Chest film taken on the day prior to left upper lobectomy showing moderate egree of pneumoperitoneum. *B*, Chest film taken on seventh postoperative day. Note selecve elevation of left diaphragm.

the diaphragm with the insertion of a small catheter, introduction of air beneath the diaphragm, and subsequent closure of the diaphragmatic opening by means of a purse-string suture. In spite of the advantage of selectivity offered by these methods, we do not favor introduction of pneumoperitoneum at the time of surgery or during the immediate postoperative period. We prefer the procedure now in use since it allows proper evaluation of the patient's tolerance of pneumoperitoneum and the benefits to be expected before surgery is carried out.

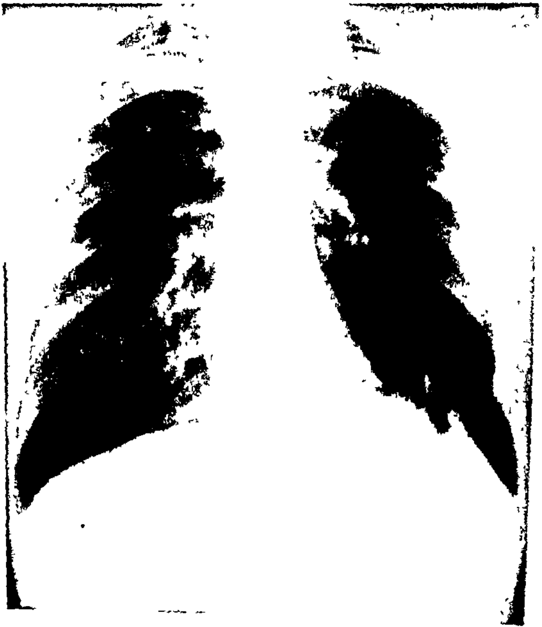


Fig. 2C.—Chest film taken eleven months after lobectomy. A pleurodiaphragmatic adhesion remains but the diaphragm functions well.

Some of our associates have objected to the use of pneumoperitoneum in this manner, with claims that the presence of air in the peritoneal cavity and associated elevation of the diaphragms renders surgery technically more difficult and contributes to prolonged and irregular anesthesia. We can only state that our experiences have failed thus far to confirm these objections in a single instance.

SUMMARY

1. The value of lobectomy and segmental resection in the treatment of tuberculosis has been clearly established, but the problem of dealing with the residual space resulting from removal of pulmonary tissue continues to tax the ingenuity of the thoracic surgeon.

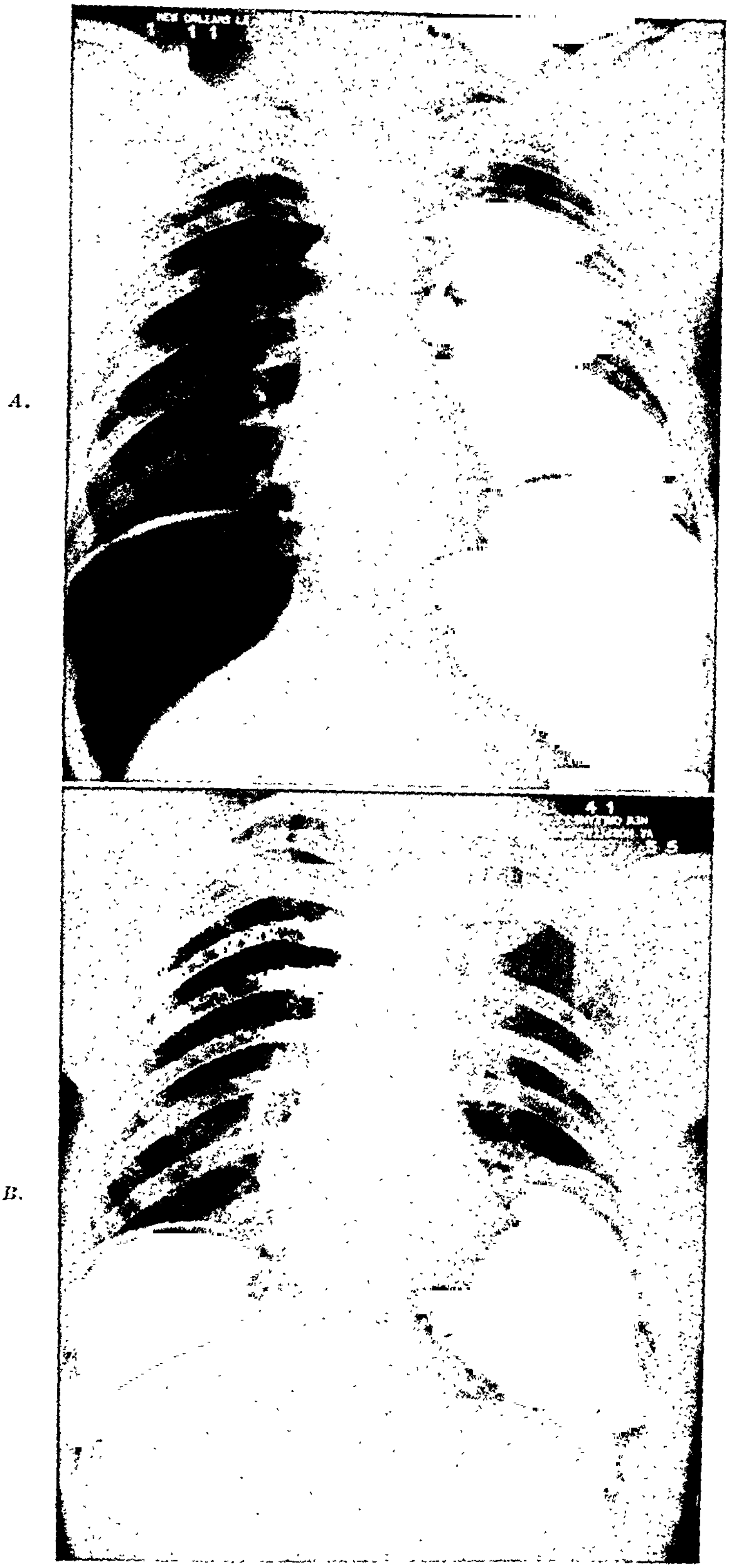


Fig. 2.—*A*, Chest film taken on the day prior to left upper lobectomy showing moderate degree of pneumoperitoneum. *B*, Chest film taken on seventh postoperative day. Note selective elevation of left diaphragm.

# LEIOMYOMA OF TRACHEA

## Case Report

Allen E Greer, M D, and George L Winn, M D
Oklahoma City, Okla

PRIMARY benign endotracheal tumors are rare neoplasms. The successful treatment of a patient with a leiomyoma of the distal trachea which had masqueraded as bronchial asthma for three years is of sufficient interest to warrant the report of a single case.

A search of the literature revealed few endotracheal or endobronchial tumors of smooth muscle origin. Langston,[1] in a collective review of [illegible] bronchial tumors, reported two such neoplasms in the Army Institute of Pathology. Unger[2,3] reported a tumor of the trachea which was classified as a leiomyoma. This was removed locally on several occasions by Dr Jerome Head and later resected with the upper lobe. A plastic reconstruction was performed by suturing the bronchus intermedius to the trachea. Dr Head[4] has also performed a pneumonectomy on another patient upon whom a lower lobe lobectomy had been performed for a papillary leiomyoma. This had extended after the initial surgery and necessitated complete removal of the lung. Following this procedure there was tracheal involvement with eventual death from suffocation. A review of tracheal tumors by Gilbert and his colleagues[5] reported only two leiomyomas of the trachea. Hirose and Hennigar reported a case of intrabronchial leiomyoma found at autopsy; they reviewed the literature and found only two intrabronchial tumors of this type.[6] Numerous fibrous tumors are reported and, perhaps, if a careful pathologic study were made of these neoplasms some of them would be classified as leiomyomas. Sweet and co-workers[7] pointed out the difficulty in pathologic classification of leiomyomas of the esophagus and the verification of such tumors reported in the literature. This difficulty in pathologic diagnosis is well known.

### CASE REPORT

A white man, aged 56, was admitted to St Anthony Hospital for treatment in December, 1951. The history revealed he had had onset of a nonproductive cough following a severe common cold six years prior to admission. About three years after onset, the cough became productive of mucoid sputum and there was the appe[illegible] f [illegible] ea [illegible] The latter seemed rather typical of asthma in that it was paroxysmal, [illegible] night, and was relieved by sitting. There was expectoration of thick, te[illegible] sputum. He was treated with adrenaline by inhalation and injection [illegible] sion, he [illegible] tory

One and one half months pr[illegible] on of fever, increased dyspnea, cough, [illegible]

---

Received for publication [illegible]

2. None of the commonly employed space-filling procedures have proved to be fully satisfactory, since each is associated with significant disadvantages.

3. Pneumoperitoneum appears to function very efficiently in this respect, but its value seems to have been largely overlooked. When compared to other types of space-filling methods such as thoracoplasty, plombage operations, and phrenic crush, the advantages of pneumoperitoneum become obvious. It produces no deformity, does not require additional surgery, is not associated with significant complications, causes no important reduction of pulmonary function, usually produces selective elevation of the diaphragm on the side operated upon, is a completely reversible procedure, and allows gradual subsequent relaxation of the remaining lung.

4. Although the present series is small, early favorable results certainly justify continued use of pneumoperitoneum for this purpose.

## REFERENCES

1. Chamberlain, J. M., Storey, C. F., Klopstock, R., and Daniels, C. F.: Segmental Resection for Pulmonary Tuberculosis, J. THORACIC SURG. 26: 471-485, 1953.
2. Gaensler, E. A., and Strieder, J. W.: Progressive Changes in Pulmonary Function After Pneumonectomy: The Influence of Thoracoplasty, Pneumothorax, Oleothorax, and Plastic Sponge Plombage on the Side of Pneumonectomy, J. THORACIC SURG. 22: 1-34, 1951.
3. Woodruff, W., Merkel, C. G., and Wright, G. W.: Decisions in Thoracic Surgery as Influenced by the Knowledge of Pulmonary Physiology, J. THORACIC SURG. 26: 156-183, 1953.
4. Taylor, W. J., Petter, J. B., and Whittenberger, J. L.: Pulmonary Function Before and After Resection of Tuberculous Lung Segments, Am. Rev. Tuberc. 72: 453-464, Oct. 1955.
5. Chamberlain, J. M., and Klopstock, R.: Further Experiences With Segmental Resection in Tuberculosis, J. THORACIC SURG. 20: 843-853, 1950.
6. Chamberlain, J. M., and Ryan, T. C.: Segmental Resection in Pulmonary Diseases, J. THORACIC SURG. 19: 199-206, 1950.

Goldman, A., Preuss, F., Gyarfas, W., Abrahams, J., Rosner, B., and Rogers, A. E. T.: Segmental Resection for Pulmonary Tuberculosis, Am. Rev. Tuberc. 70: 285-295, 1954.

Bickford, B. J., Edwards, F. R., Esplen, J. R., Gifford, J. H., Mair, A. M., and Thomas, O. F.: Lung Resection for Pulmonary Tuberculosis, Thorax 6: 25-42, 1951.

a fair response to antibiotic therapy, epinephrine, oxygen, and ACTH therapy. He was referred by his local physician, because it was felt there was a suppurative process or some other pathologic condition underlying the asthma.

The past history revealed that he had had a bronchogram made three years prior to the admission and it was said to have shown some bronchiectasis of the right lower lobe (Fig. 1). A review of these bronchograms showed the tumor well demonstrated; however, these were not obtained until after treatment had been carried out.

Examination of the patient revealed moderate respiratory distress, both inspiratory and expiratory, but chiefly the latter. Dry sibilant râles were heard bilaterally without any localizing signs. There was much mucoid sputum raised which seemed to give temporary relief.

Roentgenograms of the chest and fluoroscopy revealed the lung markings to be increased in the right base posteriorly, and some thickening of the right hilum with apparent pulsation upon fluoroscopy which was thought to be the pulmonary artery. Inspiration and expiration films were of no aid (Fig. 2).

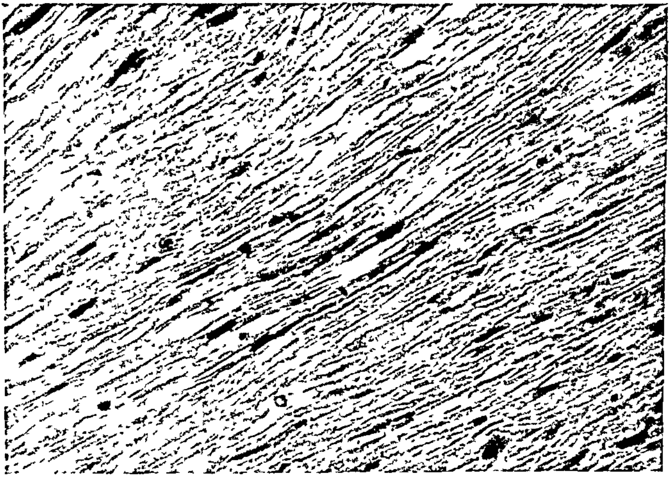


Fig. 3—Photomicrograph demonstrates the well-differentiated smooth muscle cells arranged in bundles with a whirled appearance Some of the nuclei appear large, but no mitotic figures were seen

The patient responded to epinephrine, sedation, oxygen, potassium iodide, and intravenous glucose therapy, and was discharged to be followed by the allergist. He failed to continue to do well on these measures, however. There were repeated milder attacks of dyspnea, and the cough persisted even when there was no dyspnea. He was admitted again to the hospital on Jan. 6, 1953.

Bronchoscopy revealed a pink, smooth, slightly lobulated tumor in the distal trachea, almost occluding the entire lumen at the bifurcation. There was only a small air passage to both main-stem bronchi anterior to the tumor. The biopsy was reported as demonstrating a benign tumor. Piecemeal removal of the tumor was attempted through the bronchoscope, and about one third of the tumor was removed in this fashion. There was

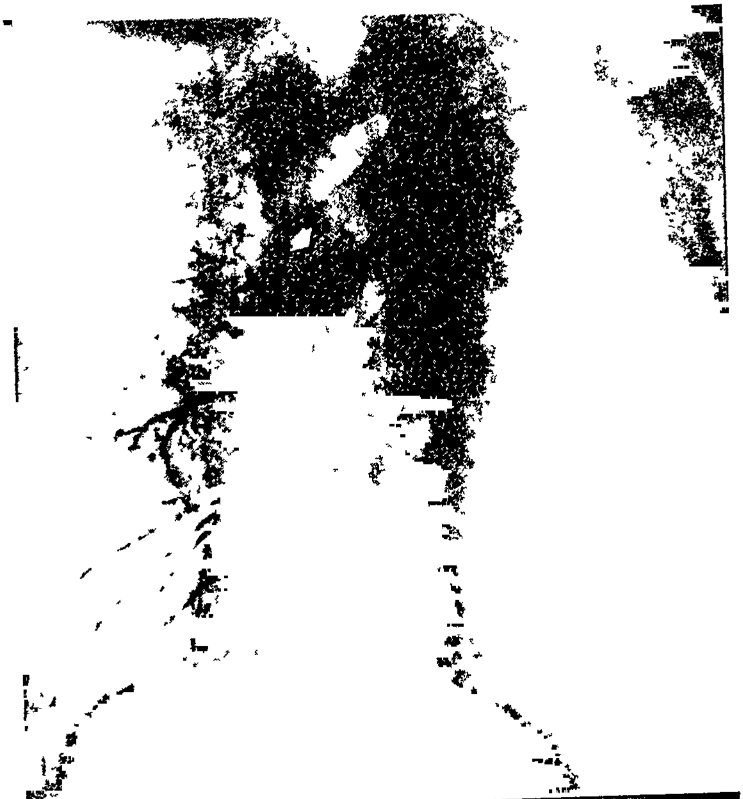


Fig. 1.

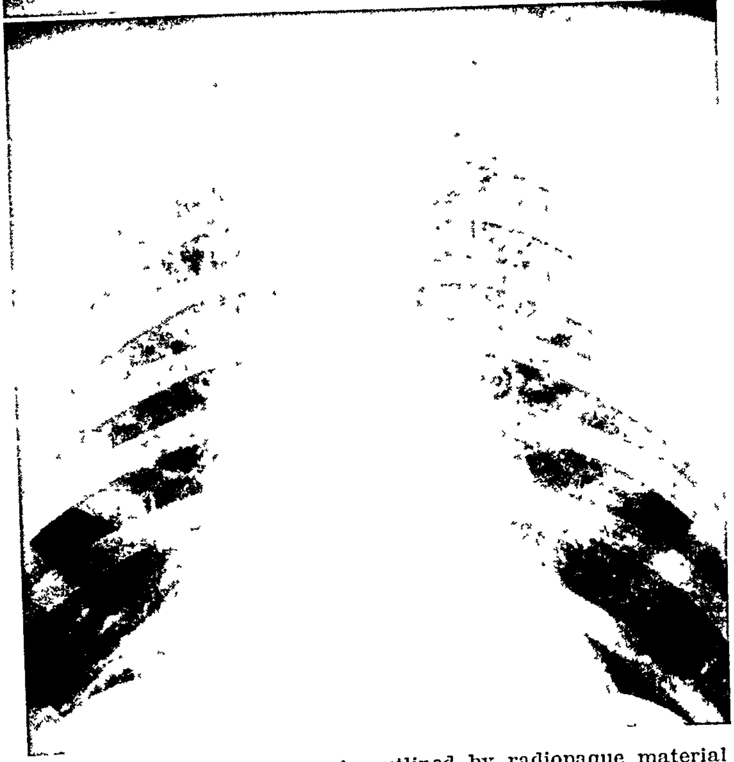


Fig. 2.

Fig. 1.—Rounded endotracheal tumor is outlined by radiopaque material in this obliqu view of bronchogram.

Fig. 2.—Inspiratory posteroanterior view of chest revealed slight increase in lung markings in the right base and slight enlargement of the right hilum

arising in the trachea are not visualized at all upon routine posteroanterior views, as is well demonstrated by this reported case. Bronchography may be a valuable adjunct because the tumor mass may show readily with this double contrast technique Planigrams and Bucky films are helpful but were not used in this instance.

Treatment consists of removal of the tumor, either by endoscopic piecemeal removal or by tracheotomy. In the reported case, the firm nature of the tissue precluded removal of sufficient amount to be safe, and second, because *the rather wide base of origin from the mucosa was not visible due to the* size of the tumor, it was not possible to attack this area of origin directly. A resectoscope may be useful in these instances

Bronchoscopy or tracheotomy through the membranous portion of the bronchus or trachea is easily performed and was the method chosen, as sufficient tissue could not be removed piecemeal without danger of a respiratory accident In case of either benign or malignant intrathoracic tracheal tumors, where the entire thickness of the tracheal wall may be involved and must be removed, plastic repair is possible as evidenced by successful cases reported by Clagett, Moersch and Grindlay,[8] and repair of stenotic lesions reported by Gebauer [9]

## SUMMARY

An endotracheal leiomyoma caused symptoms suggesting asthma The diagnosis of tumor of the distal trachea was made by bronchoscopy and removal was performed by thoracotomy and tracheotomy

## REFERENCES

1 Langston, H T: *Collective Review. Benign Endobronchial Tumors, Int Abst Surg* 91: 521, 1950.
2 Unger, L The Recognition of Non Allergic Asthma, Dis Chest 22 671, 1952
3 Unger, L Personal communication
4 Head, J: Personal communication
5 Gilbert, J D, Mazzarella, L A, and Feit, L J Primary Tracheal Tumors in the Infant and Adult, A M A Arch Otolaryng 58 1, 1953
6 Hirose, F M, and Hennigar, G R Intrabronchial Leiomyoma, J THORACIC SURG 29. 502, 1955
7 Sweet, Richard H, Soutter, L, Valenzuela, C T Muscle Wall Tumors of the Esophagus, J THORACIC SURG 27 13, 1954
8 Clagett, O T, Moersch, H J, and Grindlay, J H Intrathoracic Tracheal Tumors Development of Surgical Technics for Their Removal, Ann Surg 136 520, 1952
9 Gebauer, Paul W: Bronchial Resection and Anastomosis, J THORACIC SURG 26: 241, 1953

moderate bleeding with this manipulation, and more could not be done at this time because of the firm nature of the tumor. However, an airway was established along the left side, and it was then evident that the tumor arose from the membranous portion of the trachea in an area about 1.5 cm. in diameter.

About fourteen hours later, the patient had a severe attack of dyspnea, cyanosis, and loss of consciousness for a few seconds. Moderate dyspnea continued, the cause of which was not apparent. When a bronchoscope was introduced, there appeared to be a larger airway present than prior to the piecemeal removal. Enough tumor had probably been removed to allow the remainder to rotate upward into the trachea and thus occlude more airway than in its former position when confined to the bifurcation.

A long endotracheal tube was passed into the left bronchus, and a right thoracotomy was performed under general anesthesia. The tumor was removed by a longitudinal incision in the membranous portion of the trachea and right main-stem bronchus. The mass was delivered and removed with the mucosa. The wall was closed with interrupted sutures of No. 3-0 black silk and no air leak was evident with positive pressure. The chest was closed in the usual fashion with two thoracotomy tubes. A tracheotomy was *performed at the termination of the operation.*

Convalescence was uneventful, and there have been no subsequent asthmatic symptoms. Bronchoscopy, performed three months postoperatively, revealed no abnormality of the trachea and no evidence of the previous surgery. He is asymptomatic three and one-half years following surgery.

The tumor was comprised of well-differentiated, smooth muscle cells, arranged in bundles, which had a whirled appearance. Some of the nuclei were large; however, no mitotic figures were seen. In most areas the nuclei were separated by the fibers of the smooth muscles, which produced a dense rather than cellular appearance. Portions were covered by respiratory epithelium and there were some areas of squamous-cell metaplasia. A Weigert-Van Gieson stain showed most of the tissue to be stained yellow (smooth muscle). A few small strands of red-stained tissue were present throughout the section (connective tissue) (Fig. 3).

Sections were sent to Dr. J. B. Hazard, Cleveland Clinic Foundation, Cleveland, Ohio, and he was in agreement with the diagnosis of leiomyoma of the trachea.

## DISCUSSION

The symptom of cough, which preceded by two years the onset of wheezing and dyspnea, and persisted, despite periods of relief, pointed to the necessity of a diagnostic bronchoscopy. Clagett, Moersch, and Grindlay[8] have emphasized that recent onset of wheezing in a patient more than 40 years of age should not be considered to be due to bronchial asthma until the possible presence of an obstructing lesion of the trachea has been excluded. The symptoms are dependent upon the obstructing nature of the lesion, ulceration, and secondary infection. Cough is a common symptom and very often is dry or productive of mucoid sputum. At times the sputum is purulent due to secondary infection or blood streaked secondary to ulceration. Dyspnea and wheezing are rather constant symptoms and may be intermittent or constant. These two latter symptoms frequently lead to a mistaken diagnosis of asthma for obstructing lesions of the trachea.

Endotracheal tumor may be evident by roentgenologic study but absolute diagnosis is dependent upon bronchoscopy and tissue study. Special x-rays such as a lateral view of the trachea or overexposed posteroanterior films may demonstrate an opaque lesion in the air-filled air passages. Often tumors arising in the trachea are not visualized at all upon routine posteroanterior views, as is well demonstrated by this reported case. Bronchography may be a valuable adjunct because the tumor mass may show readily with this double contrast technique. Planigrams and Bucky films are helpful but were not used in this instance.

Treatment consists of removal of the tumor, either by endoscopic piecemeal removal or by tracheotomy. In the reported case, the firm nature of the tissue precluded removal of sufficient amount to be safe, and second, because the rather wide base of origin from the mucosa was not visible due to the size of the tumor, it was not possible to attack this area of origin directly. A resectoscope may be useful in these instances.

Bronchoscopy or tracheotomy through the membranous portion of the bronchus or trachea is easily performed and was the method chosen, as sufficient tissue could not be removed piecemeal without danger of a respiratory accident. In case of either benign or malignant intrathoracic tracheal tumors, where the entire thickness of the tracheal wall may be involved and must be removed, plastic repair is possible as evidenced by successful cases reported by Clagett, Moersch and Grindlay,[8] and repair of stenotic lesions reported by Gebauer.[9]

## SUMMARY

An endotracheal leiomyoma caused symptoms suggesting asthma. The diagnosis of tumor of the distal trachea was made by bronchoscopy and removal was performed by thoracotomy and tracheotomy.

## REFERENCES

1. Langston, H. T.: Collective Review: Benign Endobronchial Tumors, Int. Abst. Surg. 91: 521, 1950.
2. Unger, L.: The Recognition of Non Allergic Asthma, Dis. Chest 22: 671, 1952.
3. Unger, L.: Personal communication.
4. Head, J.: Personal communication.
5. Gilbert, J. D., Mazzarella, L. A., and Feit, L. J.: Primary Tracheal Tumors in the Infant and Adult, A. M. A. Arch. Otolaryng. 58: 1, 1953.
6. Hirose, F. M., and Hennigar, G. R.: Intrabronchial Leiomyoma, J. THORACIC SURG. 29: 502, 1955.
7. Sweet, Richard H., Soutter, L., Valenzuela, C. T.: Muscle Wall Tumors of the Esophagus, J. THORACIC SURG. 27: 13, 1954.
8. Clagett, O. T., Moersch, H. J., and Grindlay, J. H.: Intrathoracic Tracheal Tumors: Development of Surgical Technics for Their Removal, Ann. Surg. 136: 520, 1952.
9. Gebauer, Paul W.: Bronchial Resection and Anastomosis, J. THORACIC SURG. 26: 241, 1953.

moderate bleeding with this manipulation, and more could not be done at this time because of the firm nature of the tumor. However, an airway was established along the left side, and it was then evident that the tumor arose from the membranous portion of the trachea in an area about 1.5 cm. in diameter.

About fourteen hours later, the patient had a severe attack of dyspnea, cyanosis, and loss of consciousness for a few seconds. Moderate dyspnea continued, the cause of which was not apparent. When a bronchoscope was introduced, there appeared to be a larger airway present than prior to the piecemeal removal. Enough tumor had probably been removed to allow the remainder to rotate upward into the trachea and thus occlude more airway than in its former position when confined to the bifurcation.

A long endotracheal tube was passed into the left bronchus, and a right thoracotomy was performed under general anesthesia. The tumor was removed by a longitudinal incision in the membranous portion of the trachea and right main-stem bronchus. The mass was delivered and removed with the mucosa. The wall was closed with interrupted sutures of No. 3-0 black silk and no air leak was evident with positive pressure. The chest was closed in the usual fashion with two thoracotomy tubes. A tracheotomy was performed at the termination of the operation.

Convalescence was uneventful, and there have been no subsequent asthmatic symptoms. Bronchoscopy, performed three months postoperatively, revealed no abnormality of the trachea and no evidence of the previous surgery. He is asymptomatic three and one-half years following surgery.

The tumor was comprised of well-differentiated, smooth muscle cells, arranged in bundles, which had a whirled appearance. Some of the nuclei were large; however, no mitotic figures were seen. In most areas the nuclei were separated by the fibers of the smooth muscles, which produced a dense rather than cellular appearance. Portions were covered by respiratory epithelium and there were some areas of squamous-cell metaplasia. A Weigert-Van Gieson stain showed most of the tissue to be stained yellow (smooth muscle). A few small strands of red-stained tissue were present throughout the section (connective tissue) (Fig. 3).

Sections were sent to Dr. J. B. Hazard, Cleveland Clinic Foundation, Cleveland, Ohio, ad he was in agreement with the diagnosis of leiomyoma of the trachea.

## DISCUSSION

The symptom of cough, which preceded by two years the onset of wheezig and dyspnea, and persisted, despite periods of relief, pointed to the ecessity of a diagnostic bronchoscopy. Clagett, Moersch, and Grindlay[8] have emphasized that recent onset of wheezing in a patient more than 40 years of age should not be considered to be due to bronchial asthma until the possible presence of an obstructing lesion of the trachea has been excluded. The symptoms are dependent upon the obstructing nature of the lesion, ulceration, and secondary infection. Cough is a common symptom and very often is dry or productive of mucoid sputum. At times the sputum is purulent due to secondary infection or blood streaked secondary to ulceration. Dyspnea and wheezing are rather constant symptoms and may be intermittent or constant. These two latter symptoms frequently lead to a mistaken diagnosis of asthma for obstructing lesions of the trachea.

Endotracheal tumor may be evident by roentgenologic study but absolute diagnosis is dependent upon bronchoscopy and tissue study. Special x-rays such as a lateral view of the trachea or overexposed posteroanterior films may demonstrate an opaque lesion in the air-filled air passages. Often tumors at the level of the 6th thoracic vertebra; it divides into right and left branches, the right one supplying branches to the oesophagus. A posterior bronchial artery arising from the 1st intercostal artery is very rarely found. *The anterior bronchial arteries* arise in part from the internal mammary artery, in part from the descending tracheal artery. Smaller branches come from the pericardial arteries and make their way into the pulmonary hilus together with mediastinal and tracheo-oesophageal arteries.''

A similar description has been given by Berry and his colleagues,[10] who also describe anterior and posterior bronchial arteries. They add that the posterior bronchial artery to the right lung may arise directly from the thoracic aorta and that to the left lung from the left sixth intercostal artery. They emphasize that the posterior bronchial arteries are much larger than the anterior.

Mathes and his co-workers[6] have given a somewhat different account as follows, "It arises in one to three trunks from the anterior side of the descending aorta, just distal to the aortic arch. Frequent variations in its origin are met with; the artery on the right commonly arises from the first intercostal artery, and occasionally from the internal mammary or subclavian artery."

Bradley[11] describes bronchial branches to the superior pulmonary lobe originating from the internal mammary artery, and two or three small bronchial arteries arising either directly from the aorta or from its first intercostal branch; these latter bronchial arteries supply also the bronchial lymph nodes and the esophagus.

Miller[12] describes the bronchial arteries in the following manner, "The oesophageal and bronchial arteries vary in number and origin. Usually, the chief supply to the lungs is an artery which leaves the right sixth (rarely seventh) intercostal artery, close to its origin. This is the small '*bronco oesophageal artery.*' It crosses the left face of the oesophagus and contributes a branch to it, the oesophageal artery. It terminates shortly afterwards in right and left bronchial arteries which supply the lymph nodes and the connective tissue of the root of the lungs. The oesophagus always receives branches from some of the right intercostal arteries, caudal to the sixth. The lungs frequently receive additional branches from adjacent intercostals also."

Bourdelle and Bressou[13] state that "a bronchial artery" arises from the thoracic aorta and proceeds from there to the bifurcation of the trachea to be distributed in the lung.

## MATERIALS AND METHODS

The present work is based on the dissection of 30 dogs, male and female, of different breeds and of various ages. The bronchial arteries of these dogs were filled with colored latex; injection of the latex, in some instances, was made into the femoral or common carotid artery and in others directly into the aorta or one of its branches. In some cases, the thorax was opened prior to injection so that the lungs retracted; in other cases, the thorax
closed. No difference in the filling of the arteries was observed in

# THE ANATOMY OF THE BRONCHIAL ARTERIES OF THE DOG

HILEL NOTKOVICH, M.D.
JERUSALEM, ISRAEL

THE importance of the bronchial arteries in the pulmonary blood circulation has been repeatedly observed since they were first described by Ruysch[1] and Haller[2]; likewise their participation in various vascular and parenchymatous pathologic processes of the lungs has been noted, whether these be of natural origin or experimentally produced. The subject has been well reviewed by Cauldwell,[3] Liebow[4] and their colleagues, and Tobin and Zariquiey.[5]

Where knowledge concerning the bronchial arteries has been gained from experimental procedures, these experiments have usually been carried out on the other vessels of the lung, the effects upon the bronchial arteries or the responses on their part being thereafter observed. Very few experiments have been based upon direct operative intervention on the bronchial arteries. This is mainly due to technical difficulties which result from their small caliber, and the great number of branches that enter the lung, as well as from their rich anastomoses and intimate relations with the other structures of the pulmonary pedicle. Thus, Mathes and his co-workers,[6] after trying to ligate the bronchial arteries of the dog, wrote as follows, "The isolation and ligation of the entire bronchial arteries was found to be impossible after several attempts. The main arteries arising from the aorta were tied, but in each case after autopsy the lung was entirely normal and the bronchial artery injected in its entirety from the bronchial vessels arising from the intercostal artery." More recently Tobin and Zariquiey[5] have stated, "The tortuous course and the location of the bronchial arteries, however, make it almost a physical impossibility to occlude them directly without closure of the bronchial lumen."

The present work was undertaken to acquire a more exact knowledge of the topographical anatomy of the bronchial arteries of the dog, particularly as regards their origin and their mediastinal course; it was considered that such knowledge might well facilitate operative procedures upon these vessels.

Although complete and detailed descriptions of the topographical anatomy of the bronchial arteries in man have been made by several authors,[3, 7, 8] the topographical anatomy of the bronchial arteries in the dog has been inadequately dealt with. Detailed descriptions are lacking from current literature, as well as from texts on the anatomy of the dog.

Küttner[9] described the bronchial arteries of the dog as follows: "*The posterior bronchial artery* arises by a common trunk with the intercostal artery

---

From the Department of Anatomy, The Hebrew University—Hadassah Medical School, Jerusalem, Israel.
Received for publication Jan. 18, 1956.

For convenience the cases were grouped as follows (Table I):

Type I: 1 right and 1 left bronchial artery
Type II: 1 right and 2 left bronchial arteries
Type III: 2 right and 1 left bronchial artery
Type IV: 2 right and 2 left bronchial arteries.

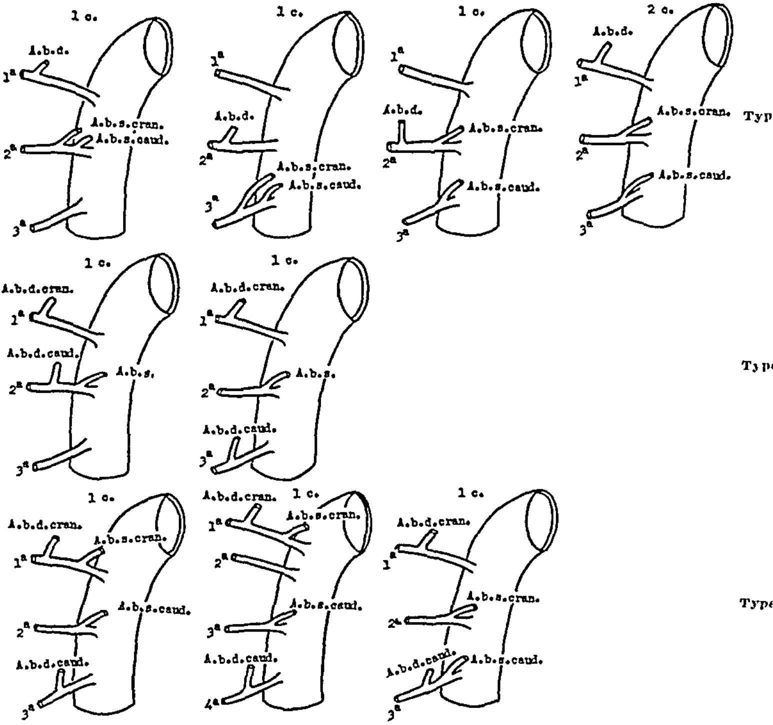


Fig. 1.—Cont'd. Type II. (16.6 per cent). One right and two left bronchial arteries
Type III. (6.6 per cent). Two right and one left bronchial arteries
Type IV. (10 per cent). Two right and two left bronchial arteries

Fig. 1 shows schematically all the possible variations of origin and the frequency with which they were met. It will be seen that Type I was by far the most common.

TABLE I. CLASSIFICATION OF CASES ACCORDING TO NUMBER OF BRONCHIAL ARTERIES OF AORTIC INTERCOSTAL ORIGIN TO EACH SIDE

| TYPE | BRONCHIAL ARTERY | | NUMBER OF CASES | |
|---|---|---|---|---|
| | RIGHT | LEFT | | |
| I | 1 | 1 | | |
| II | 1 | 2 | | |
| III | 2 | 1 | | |
| IV | 2 | 2 | | |

two methods nor did artificial inflation of the lungs via the trachea affect the filling. In those cases where the thorax was already open at the time of injection, the aorta was ligated at its exit from the heart so as to prevent the injection material from flowing backward into the pulmonary veins.

The latex injections were made either with a veterinary syringe having a rubber piston or by means of an air-compressor like that used for tree-spraying. The compressor, with its manometer, is connected by rubber-tubing to a flask containing the latex; a second tube emerging from the flask is connected to the injection needle. This apparatus proved to be most practical.

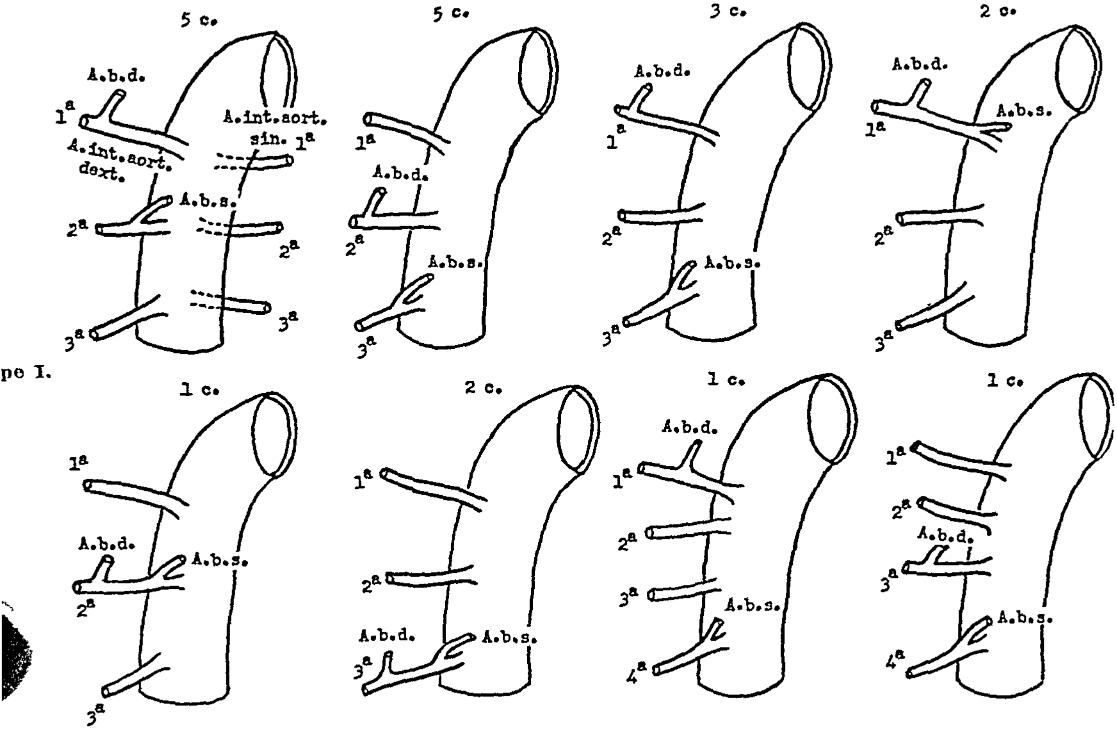


Fig. 1.—Schematic representation of the variations in the origins of the bronchial arteries from the aortic intercostal arteries.

Type I.—(66.6 per cent). One right and one left bronchial artery.
A.int.aort.dext.: Arteria intercostalis aortica dextra.
A.int.aort.sin.: Arteria intercostalis aortica sinistra.
A.b.d.cran.: Arteria bronchialis dextra cranialis.
A.b.s.cran.: Arteria bronchialis sinistra cranialis.
A.b.d.caud.: Arteria bronchialis dextra caudalis.
A.b.s.caud.: Arteria bronchialis sinistra caudalis.
1 c. to 5 c. represent the number of cases observed.

After fixation of the parts in formalin for a few days, the sternum in each case was removed and also the greater part of the ribs to within a few centimeters of the vertebral column. On drawing the lungs ventrally, the ramifications of the injected bronchial arteries were clearly observed through the transparent parietal pleura and were then dissected toward their origins and terminations.

## OBSERVATIONS AND DESCRIPTION

One or two bronchial arteries were found going to each lung. Where two arteries are present on one side they are referred to in the present paper as the cranial and caudal bronchial arteries (right or left), respectively.

costal spaces to which they are distributed (Fig. 2). Thus, it may be noted from Table III that the vertebral levels of the points of origin of the bronchial arteries extend from the disc between the fourth and the fifth thoracic vertebra down to the eighth thoracic vertebra. The majority of the right bronchial arteries have their origin in front of the fifth and sixth thoracic vertebrae and the disc between them, while most left bronchial arteries arise in front of the sixth and seventh thoracic vertebrae and the disc between them.

Table III. Vertebral Level of Origin of the Bronchial Arteries From the Aortic Intercostal Arteries

| VERTEBRAL LEVEL | RIGHT BRONCHIAL ARTERY | | | LEFT BRONCHIAL ARTERY | | | TOTAL |
|---|---|---|---|---|---|---|---|
| | | DOUBLE | | | DOUBLE | | |
| | SINGLE | CRANIAL | CAUDAL | SINGLE | CRANIAL | CAUDAL | |
| $T_4$ | - | - | - | - | - | - | - |
| Disc $T_4$ $T_5$ | 2 | - | - | - | 1 | - | 3 |
| $T_5$ | 7 | 2 | - | 2 | 1 | 1 | 13 |
| Disc $T_5$ $T_6$ | 6 | 2 | - | 2 | 1 | 1 | 12 |
| $T_6$ | 9 | 1 | 3 | 10 | 1 | 2 | 29 |
| Disc $T_6$ $T_7$ | - | - | - | 1 | 1 | 1 | 3 |
| $T_7$ | 1 | - | 1 | 7 | - | 3 | 12 |
| Disc $T_7$ $T_8$ | - | | - | - | - | - | - |
| $T_8$ | - | - | 1 | - | - | - | 1 |

Disc = Intervertebral disc
T = Thoracic vertebra

The aortic intercostal arteries, giving origin to the bronchial arteries, spring from the right lateral aspect of the descending aorta, sometimes close to its ventral aspect or even from the right lateral portion of the ventral aspect itself Very often these intercostals have a greater caliber than the remaining intercostal arteries

*Course* (Figs 1-4) —

*Right bronchial arteries:*

*A. Single right bronchial artery:* This is the most constant of the bronchial arteries in its origin, course, and distribution It springs from the first, second, or third right intercostal artery 10 to 25 mm from the aorta, it passes upward and forward, lying first on the right side of the vertebral column, then, crossing the right aspect of the esophagus, it reaches the trachea, descends upon the right main bronchus and divides into its various branches which enter the lung with that bronchus At the level of the trachea, the right bronchial artery crosses the vagus nerve on its lateral side or it intermingles with the various branches into which the vagus nerve has divided at that level On the whole, the artery describes an arch open downward and

*Origin.*—

The bronchial arteries of *both* sides were found to arise from the *right* aortic intercostal arteries. In no case were they found arising directly from the aorta or from the left intercostal arteries.

The point of origin from the intercostal arteries differs according to the bronchial artery in question; it was found at a distance of 0.5 mm. to 25 mm. from the aorta.

The right bronchial arteries generally arise from the intercostal arteries considerably farther from the aorta than the left ones; the cranial bronchial arteries arise farther away from the aorta than the caudal ones. Thus, the right cranial artery arises farthest from the aorta, and the left caudal rises nearest the aorta (Fig. 1). When the point of origin of a bronchial artery is very close to the aorta, that is, when there is a very short common trunk of origin from the bronchial and intercostal arteries (i.e., a very short "intercostobronchial artery"[3]), it may at first sight appear that the bronchial artery arises directly from the aorta, but by close observation its origin from the intercostal artery may be distinguished. This often happens with the left caudal bronchial artery.

The particular right *aortic* intercostal arteries from which a bronchial artery may spring are the first to the fourth. The frequency with which the different bronchial arteries arise from these intercostal arteries is shown in Table II, where it may be seen that most right bronchial arteries arise from the first or second aortic intercostal arteries, while the left bronchial arteries arise most commonly from the second or third aortic intercostal.

ABLE II. FREQUENCY OF ORIGINS OF BRONCHIAL ARTERIES FROM AORTIC INTERCOSTAL ARTERIES

| RIGHT AORTIC INTER-COSTAL ARTERY | RIGHT BRONCHIAL ARTERIES | | | LEFT BRONCHIAL ARTERIES | | | TOTAL |
|---|---|---|---|---|---|---|---|
| | | DOUBLE | | | DOUBLE | | |
| | SINGLE | CRANIAL | CAUDAL | SINGLE | CRANIAL | CAUDAL | |
| First | 14 | 5 | - | 2 | 2 | - | 23 |
| Second | 8 | - | 1 | 8 | 5 | 2 | 24 |
| Third | 3 | - | 3 | 10 | 1 | 6 | 23 |
| Fourth | - | - | 1 | 2 | - | - | 3 |

It is to be noted that the numbering of the aortic intercostal arteries does ot correspond to that of the intercostal spaces. A variable number (1 to 4) pper intercostal spaces are supplied by the costocervical trunk of the subavian artery. The aortic intercostal arteries from which the bronchial rteries arise (i.e., first to fourth) pass to the third to eighth intercostal paces; one aortic intercostal artery may supply more than one intercostal pace.

It is convenient also to relate the points of origin of the bronchial rteries (from the intercostal arteries) to vertebral levels. Because of the oblique course taken by the intercostal arteries, they, i.e., the intercostal arteries, may arise from the aorta at a level higher or lower than the intercostal spaces to which they are distributed (Fig. 2). Thus, it may be noted from Table III that the vertebral levels of the points of origin of the bronchial arteries extend from the disc between the fourth and the fifth thoracic vertebra down to the eighth thoracic vertebra. The majority of the right bronchial arteries have their origin in front of the fifth and sixth thoracic vertebrae and the disc between them, while most left bronchial arteries arise in front of the sixth and seventh thoracic vertebrae and the disc between them.

Table III. Vertebral Level of Origin of the Bronchial Arteries From the Aortic Intercostal Arteries

| VERTEBRAL LEVEL | RIGHT BRONCHIAL ARTERY | | | LEFT BRONCHIAL ARTERY | | | TOTAL |
|---|---|---|---|---|---|---|---|
| | | DOUBLE | | | DOUBLE | | |
| | SINGLE | CRANIAL | CAUDAL | SINGLE | CRANIAL | CAUDAL | |
| $T_4$ | - | - | - | - | - | - | - |
| Disc $T_4$-$T_5$ | 2 | - | - | - | 1 | - | 3 |
| $T_5$ | 7 | 2 | - | 2 | 1 | 1 | 13 |
| Disc $T_5$-$T_6$ | 6 | 2 | - | 2 | 1 | 1 | 12 |
| $T_6$ | 9 | 1 | 3 | 10 | 4 | 2 | 29 |
| Disc $T_6$-$T_7$ | - | - | - | 1 | 1 | 1 | 3 |
| $T_7$ | 1 | - | 1 | 7 | - | 3 | 12 |
| Disc $T_7$-$T_8$ | - | - | - | - | - | - | - |
| $T_8$ | - | - | 1 | - | - | - | 1 |

Disc = Intervertebral disc.
T = Thoracic vertebra.

The aortic intercostal arteries, giving origin to the bronchial arteries, spring from the right lateral aspect of the descending aorta, sometimes close to its ventral aspect or even from the right lateral portion of the ventral aspect itself. Very often these intercostals have a greater caliber than the remaining intercostal arteries.

*Course* (Figs. 1-4).—

*Right bronchial arteries:*

*A. Single right bronchial artery:* This is the most constant of the bronchial arteries in its origin, course, and distribution. It springs from the first, second, or third right intercostal artery 10 to 25 mm. from the aorta, it passes upward and forward, lying first on the right side of the vertebral column, then, crossing the right aspect of the esophagus, it reaches the trachea, descends upon the right main bronchus and divides into its various branches which enter the lung with that bronchus. At the level of the trachea, the right bronchial artery crosses the vagus nerve on its lateral side or it intermingles with the various branches into which the vagus nerve has divided at that level. On the whole, the artery describes an arch open downward and

*Origin.*—

The bronchial arteries of *both* sides were found to arise from the *right* aortic intercostal arteries. In no case were they found arising directly from the aorta or from the left intercostal arteries.

The point of origin from the intercostal arteries differs according to the bronchial artery in question; it was found at a distance of 0.5 mm. to 25 mm. from the aorta.

The right bronchial arteries generally arise from the intercostal arteries considerably farther from the aorta than the left ones; the cranial bronchial arteries arise farther away from the aorta than the caudal ones. Thus, the right cranial artery arises farthest from the aorta, and the left caudal rises nearest the aorta (Fig. 1). When the point of origin of a bronchial artery is very close to the aorta, that is, when there is a very short common trunk of origin from the bronchial and intercostal arteries (i.e., a very short "intercostobronchial artery"[3]), it may at first sight appear that the bronchial artery arises directly from the aorta, but by close observation its origin from the intercostal artery may be distinguished. This often happens with the left caudal bronchial artery.

The particular right *aortic* intercostal arteries from which a bronchial artery may spring are the first to the fourth. The frequency with which the different bronchial arteries arise from these intercostal arteries is shown in Table II, where it may be seen that most right bronchial arteries arise from the first or second aortic intercostal arteries, while the left bronchial arteries arise most commonly from the second or third aortic intercostal.

TABLE II. FREQUENCY OF ORIGINS OF BRONCHIAL ARTERIES FROM AORTIC INTERCOSTAL ARTERIES

| RIGHT AORTIC INTERCOSTAL ARTERY | RIGHT BRONCHIAL ARTERIES | | | LEFT BRONCHIAL ARTERIES | | | TOTAL |
|---|---|---|---|---|---|---|---|
| | | DOUBLE | | | DOUBLE | | |
| | SINGLE | CRANIAL | CAUDAL | SINGLE | CRANIAL | CAUDAL | |
| First | 14 | 5 | – | 2 | 2 | – | 23 |
| Second | 8 | – | 1 | 8 | 5 | 2 | 24 |
| Third | 3 | – | 3 | 10 | 1 | 6 | 23 |
| Fourth | – | – | 1 | 2 | – | – | 3 |

It is to be noted that the numbering of the aortic intercostal arteries does not correspond to that of the intercostal spaces. A variable number (1 to 4) upper intercostal spaces are supplied by the costocervical trunk of the subclavian artery. The aortic intercostal arteries from which the bronchial arteries arise (i.e., first to fourth) pass to the third to eighth intercostal spaces; one aortic intercostal artery may supply more than one intercostal space.

It is convenient also to relate the points of origin of the bronchial arteries (from the intercostal arteries) to vertebral levels. Because of the oblique course taken by the intercostal arteries, they, i.e., the intercostal arteries, may arise from the aorta at a level higher or lower than the inter-backward. In the first part of its course it runs nearly parallel to the arch of the azygos vein which may partially overlap the artery; in only 2 cases (6.6 per cent) was the artery seen to cross the lateral aspect of the azygos vein.

*B. Double right bronchial arteries:* 1. Where two right bronchial arteries are found, the cranial of the two, here called the *right cranial bronchial artery,* corresponds to the single right bronchial artery in its course and relations as described previously. It usually arises from the first intercostal artery.

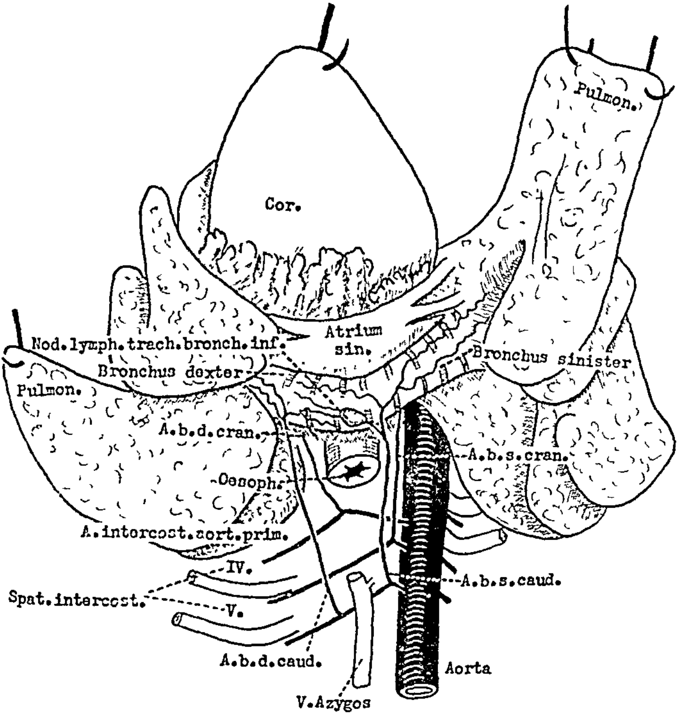


Fig. 4.—Origins and course of bronchial arteries (Type IV) as seen from the front. The heart has been drawn upward and the lungs drawn aside The bifurcation of the trachea is thus shown. The esophagus and the azygos vein have been cut.

Nod.lymph.trach.bronch inf.: Noduli lymphatici tracheo-bronchiales inferiori.

Spat intercost.: Spatium intercostale

(For key to remaining lettering see legend to Fig. 1)

2. *The right caudal bronchial artery* was seen to arise from the second, third, or fourth right aortic intercostal artery, 3 to 12 mm. from the aorta. It runs upward and forward, its direction being more vertical, the lower its. origin. It crosses the right aspect of the esophagus and reaches th. aspect of the right main bronchus, with which it descends, various branches.

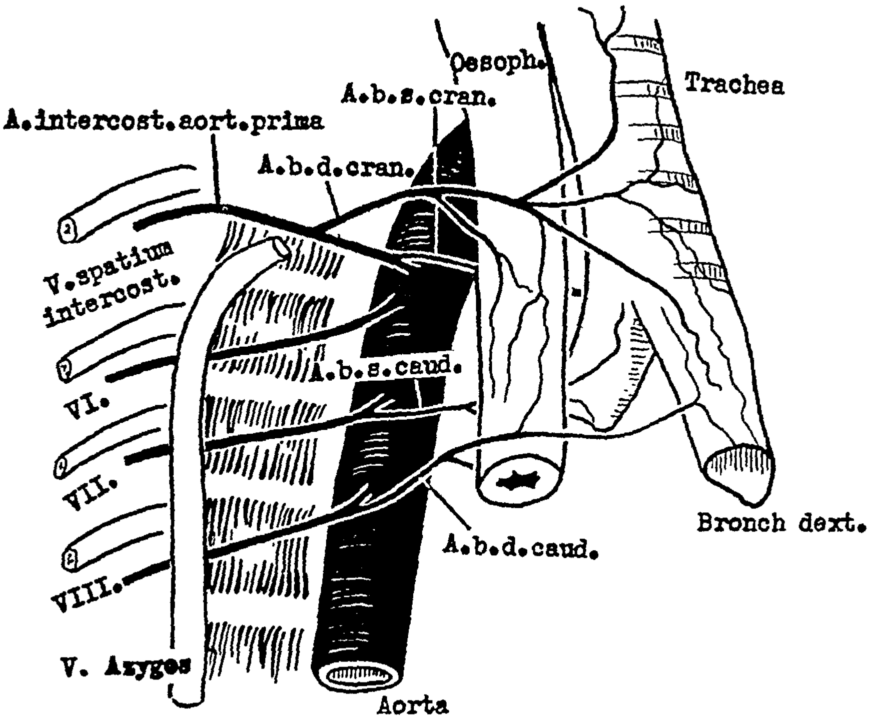


Fig. 2.

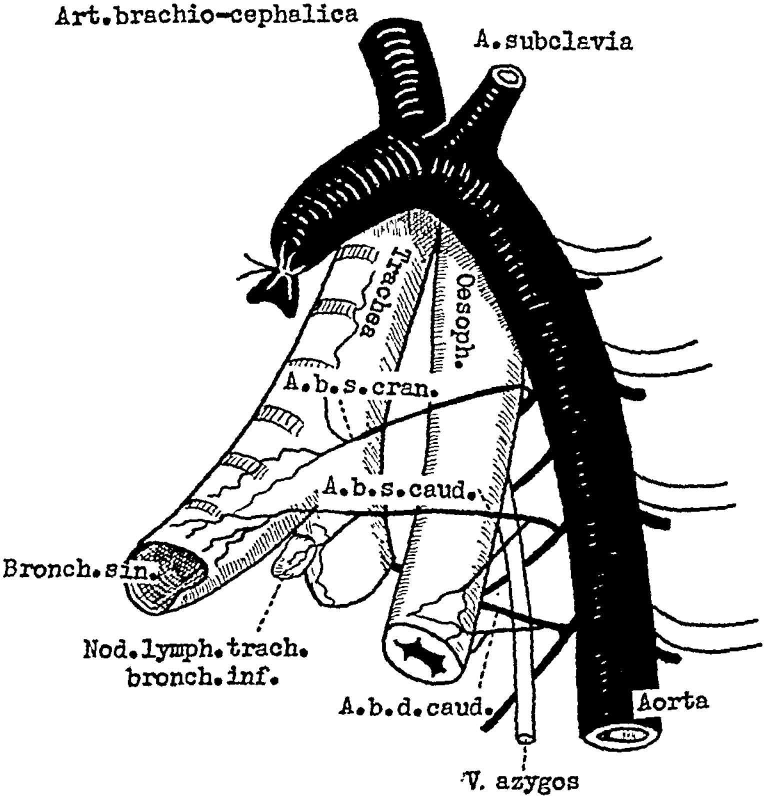


Fig. 3.

Figs. 2 and 3.—Origins and course of bronchial arteries as seen from the right (Fig. 2) and from the left (Fig. 3) in a Type IV case. In cases where a single artery only is present on one or the other side, its course is represented by the cranial of the two bronchial arteries shown in these figures. (For key to lettering see legend to Fig. 1.)

4. *Tracheal branches: to the inferior portion of the trachea.*

5. *Branches to nerves:* a. To the vagi: each bronchial artery gives off branches to the nerve of the corresponding side; these branches ascend and descend with the nerve. There are twigs accompanying the branches of the nerves to the pulmonary plexus, and on the left side branches ascend with the left recurrent laryngeal nerve. b. *Inconstant branches to the sympathetic* trunk, its ganglia and branches.

6. *Pericardial branches:* small branches to the nearby pericardium.

7. *Vasa vasorum:* from the right bronchial arteries, especially to the azygos vein and the superior vena cava; from the left bronchial arteries to the aortic arch and descending aorta. Both right and left bronchial arteries give branches to the ipsilateral pulmonary arteries and veins.

8. *Branches to lymph glands:* the lymph glands of the pulmonary pedicle and the superior tracheobronchial glands receive branches from the bronchial arteries of the corresponding side. The inferior tracheobronchial glands (i.e., glands of the tracheal bifurcation) may receive their supply from both sides or mainly from one of the bronchial arteries. As has been pointed out already, it can often be observed that these glands are sometimes pierced by branches of the bronchial arteries which pass to the opposite side.

*Anastomoses.—*

Frequent and rich anastomoses are observed between the branches of the artery of the same side, both before and after they reach the main bronchus; at the level of the tracheal bifurcation and among the inferior tracheobronchial lymph glands, one may observe fine anastomoses between the arteries of the two sides. Furthermore, by means of the branches from the *bronchial arteries to the various mediastinal organs and the parietes,* rich anastomoses are built up between the bronchial arteries and the arteries supplying those regions, e.g., innominate or brachiocephalic, subclavian, internal mammary, inferior thyroid, intercostal, thymic, diaphragmatic, and pericardial arteries. In certain cases, some anastomoses attain considerable proportions, so that the impression is created that the bronchial branches are continuations of those anastomoses; this can be observed especially on the trachea and the esophagus.

*Other Bronchial Arteries to the Lungs.—*

In one case (Type II), the right pericardiophrenic artery was seen to give branches to the right bronchus.

In one other case (Type I), a thin branch from the sixth right aortic intercostal artery which passed to the tenth intercostal space reached the inferior lobe of the lung via the pulmonary ligament.

## DISCUSSION AND CONCLUSIONS

A striking feature of the anatomy of the bronchial arteries is the asymmetry of their origin; these arteries supply bilateral organs, more or less symmetrical, yet they arise, in the main, from the intercostal arteries of the right side. Such a tendency is already observed in man,[3] where the bronchial

In some cases, small branches from one or both of the right bronchial arteries were seen to reach the left main bronchus, either directly or by crossing the inferior tracheobronchial lymphatic glands. (It will be pointed out later that, more frequently, the right main bronchus, in a similar way, receives branches from the left bronchial arteries.)

*Left bronchial arteries:*

*A. Single left bronchial artery:* This is more inconstant in its course and relations than the right arteries. It arises from the first, second, or third right aortic intercostal artery, generally from the second or third (Table II), at a distance of 0.5 to 5 mm. from the aorta. It first passes to the left, between the aorta and the esophagus, then, crossing the left aspect of the latter, it reaches the left main bronchus or it may first pass on to the trachea and then descend to the bronchus, having already divided into its branches. It crosses the left vagus nerve on its medial side or it is intermingled with the ramifications of the nerve.

*B. Double left bronchial arteries:* 1. Where two bronchial arteries are present the cranial of the two, here called the *left cranial bronchial artery,* is similar in its origin, course, and relations to the single left bronchial artery. It arises rather constantly from the second right aortic intercostal artery (Table II, Fig. 1). 2. The *left caudal bronchial artery* arises from the second or third right aortic intercostal artery, 0.5 mm. to 3 mm. from the aorta. Its course and relations are similar to those of the left superior artery, but it reaches the left main bronchus at a lower level.

Frequently the left bronchial arteries give off branches which supply the inferior tracheobronchial lymph glands and these branches continue on to the *right* main bronchus; small branches passing directly to the right bronchi were observed as well (see Figs. 3 and 4). As already noted, in a similar way, though less frequently, the right bronchial arteries give off branches to the left main bronchus. Branches of this kind are generally small and end in the proximal part of the main bronchus, but sometimes they are important and descend together with the ipsilateral bronchial arteries. In some of these cases it is justifiable to consider the bronchial arteries to the two main bronchi as arising by a common stem which ends by dividing into terminal branches for both main bronchi. Thus, the lungs may receive blood supplied to them by the bronchial arteries of both sides.

*Branches.—*

Throughout their mediastinal course, the bronchial arteries give off branches to organs and tissues in the neighborhood. The name "bronchomediastinal arteries" would fit them better because of their distribution.

Among the branches of the bronchial arteries are the following:

1. *Parietal branches:* to the prevertebral muscles and the vertebrae.
2. *Pleural branches:* to the mediastinal pleura, to the pleura of the root of the lung and to the most proximal part of the visceral pleura.
3. *Esophageal branches:* to the thoracic portion of the esophagus.

## SUMMARY

In dissections made on 30 dogs, whose arteries were previously injected with latex, one or two bronchial arteries were observed on each side It was possible to group the cases into 4 principal types

Type I 1 right and 1 left bronchial artery (66 6%)
*Type II 1 right and 2 left bronchial arteries (16 6%)*
Type III 2 right and 1 left bronchial artery (6 6%)
Type IV 2 right and 2 left bronchial arteries (10 0%)

The bronchial arteries were found in all cases to spring from the first to the fourth right aortic intercostal arteries They were never seen to arise directly from the aorta or from the left intercostal arteries Their vertebral level of origin extends from the disc between the fourth and fifth thoracic vertebrae down to the eighth thoracic vertebra The course relations, distribution, and anastomoses of the arteries are described and the best method of approach to them is outlined Other possible sources of systemic arterial supply to the lungs are pointed out

I am deeply indebted to Mr Zvi Lev of the Pedagogical Institute for Science, Jerusalem, for invaluable help rendered throughout the entire course of this work My thanks are also due to Prof M Ickowicz and Dr G Gitlin for assistance in revision of the manuscript, to Mrs L Salomon for the drawings, and to Mr Isaac Hayoun for technical assistance

## REFERENCES

1 Ruysch, F Opera omnia anatomico medico chirurgica Vol I Dilucidatio Valvularum, chapt 4, obs 15, pp 19 22, fig 9, vol 3 Epistola Anatomica Problematica Sexta (from J H Graetz), and Responsio, 1731, pp 1 11, tab 7, figs 1 5 Amstelodami Janssonio Waesbergios, 1721
2 Haller, A Icorum Anatomicarum quibus praecipuae partes corporis humani, vol 3, pp 35 37, Gottingen, 1747
3 Cauldwell, E W, Siekert, R G, Lininger, R E, and Anson, B J The Bronchial Arteries, an Anatomic Study of 150 Cadavers, Surg, Gynec & Obst 86 395 412, 1948
4 Liebow, A A, Hales, M R, Harrison, W, Bloomer, W, and Lindskog, G E The Genesis and Functional Implications of Collateral Circulation of the Lungs, Yale J Biol & Med 22 637 650, 1950
5 Tobin, Ch F, and Zariquiey, M O Some Observations on the Blood Supply of the Human Lung, M Radiog & Photog 29 9 19, 1953
6 Mathes, M E Holman E, and Reichert F L A Study of the Bronchial, Pulmonary, and Lymphatic Circulation of the Lung Under Various Pathologic Conditions Experimentally Produced, J Thoracic Surg 1 339, 1932
7 Nakamura, N Zur Anatomie der Bronchialarterien, Anat Anz 58 508 517, 1924
8 Hovelacque A, Monod, O, and Evrard H Note au sujet des artères bronchiques, Ann d'anat Path, Paris 13 129 141 1936
9 Kuttner Beitrag zur Kenntniss der Kreislaufsverhaltnisse der Saugethierlunge, Virchows arch 73 476 523, 1878
10 Berry, J L, Brailsford, J F, and de Burgh Daly, I Bronchial Vascular System in Dog, Proc Roy Soc, London 109 214 228, 1931
11 Bradley, O C Topographical Anatomy of the Dog, ed 5, Edinburgh, 1948, Oliver & Boyd, Ltd, pp 37, 51, and 73
12 Miller, L M Guide to the Dissection of the Dog, ed 2, New York, Ithaca 1949
13 Bourdelle, E, and Bressou, C Anatomie regionale des animaux domestiques IV *Carnivores chien et chat*, Paris, 1953, J B Bailliere et fils
14 Winogradew, 1929 Quoted by Hovelacque[8]

arteries spring either directly from the aorta (generally from the most right part of its anterior aspect), or from the right intercostal arteries, but never from the left intercostal arteries. It must be in very rare cases only that a bronchial artery arises from a left intercostal artery as described for the dog by Berry and his co-workers.[10] Caldwell and his colleagues[3] describe such an origin in man only in cases of situs inversus. With regard to the descriptions by others[6, 10, 11, 13] of bronchial arteries in the dog arising directly from the aorta, it is my impression that arteries which appear to arise in this manner do not in fact do so, but arise from an aortic intercostal artery close to its point of origin from the aorta.

Earlier workers[9, 10] have classified the bronchial arteries into anterior and posterior groups, the former arising from the internal mammary and its branches or from other arteries in the vicinity. In the present study, the arteries found to arise from intercostal arteries correspond with the posterior group named by these authors and they constitute the constant and most important systemic arterial supply to the lungs. As far as the anterior group is concerned, these were found in general to be small and inconstant and always merely accessory to the main supply from the posterior group.

Regarding the description of Küttner[9] and Miller,[12] that the supply to the right and left bronchi arises by a common trunk which subsequently divides to supply both sides, it was always found in the present work that separate bronchial arteries to each side were present, arising independently from aortic intercostal arteries. Frequently, however, one or another of these bronchial arteries gives branches to both sides.

As a practical application of the present anatomic description, it can be seen that the best approach to the bronchial arteries of *both* lungs in the dog is from the right side. Thus, the right hemithorax is opened and the lung drawn ventrally. On separating the esophagus from the aorta, the origin of the bronchial arteries *for both sides* may easily be localized by following the first four *aortic* intercostal arteries. The only element that overlaps the right bronchial arteries in addition to the parietal pleura is the arch of the azygos vein.

The present study contains the results of observations made on a limited number of dissections, and shows therefore only the most frequent variations in the anatomy of the bronchial arteries. It is possible that there are variations other than those just described. Thus, apart from arteries entering the lung by way of its hilum or the pulmonary ligament, branches entering any part of its surface through pleural adhesions (either naturally or experimentally produced) have been described. Furthermore, arteries of completely anomalous origin and course have been described as sometimes arising in the abdomen and perforating the diaphragm on their way to the thorax.[14]

All these possibilities of arterial blood supply, as well as the numerous and various anastomoses of the bronchial arteries with the arteries of neighboring organs, must be taken into account in a study of the systemic bronchopulmonary circulation.

TABLE II RESULTS OF RESECTION IN CASES OF PRIMARY LYMPHOSARCOMA OF THE LUNG

| AUTHOR | OPERATION | RESULT |
|---|---|---|
| Anlyan, Lovingood, and Klassen* | Right lower lobectomy | Four years after operation developed an area of increased density in right hilum which enlarged and was treated with x-ray with complete resolution 6 years after operation. No other evidence of recurrence |
| Beck and Reganis* | Right lower lobectomy | A gastric ulcer present at the time of lobectomy showed complete healing. Two years after lobectomy the ulcer recurred. Subtotal gastric resection was performed and a section of the stomach was almost replaced by lymphosarcoma. The patient is living and well with no further evidence of recurrence 5½ years after lobectomy |
| Churchill* | Right upper lobectomy | X-ray therapy postoperative. Five years later areas of opacity in both lungs treated with x-ray with marked regression. At 8½ years there is a homogeneous density in left costophrenic angles, right hilum has become larger for 2 years. At 8½ years both submaxillary glands are enlarged and are regarded clinically as lymphosarcoma |
| Ochsner and DeCamp* | Left upper lobectomy | Died 4 years after operation. No other information available |
| Rose | Right upper lobectomy | No evidence of recurrence 6 months after operation |
| Sarokhan | Left upper lobectomy | Died 5 weeks after operation due to cerebrovascular accident |
| Spitt and Grayzel* | Right lower lobectomy | Living without evidence of recurrence 14 months after operation, then lost to follow up |
| Touroff* | Right upper lobectomy | Well without evidence of recurrence 30 months after operation, then lost to follow up |
| Willis* | Right upper lobectomy | Developed empyema and was drained. Had x-ray therapy. Three years after operation had dime sized area of granulation in right bronchus which cleared. Had more x-ray therapy. At present, 12 years after operation he has right main stem bronchostenosis and some narrowing and deviation of left main stem bronchus with purulent sputum on both sides |
| Behrend* | Pneumonectomy | Well without evidence of recurrence 13 months after operation. Has since died but time and cause of death unavailable |
| Grimes* | Pneumonectomy | Well with no evidence of recurrence 4 years and 3 months after operation |
| Grimes | Pneumonectomy | Well with no evidence of recurrence 7 years after operation |
| Maier* | Pneumonectomy | Well without evidence of recurrence 10½ years after operation. A nodule appeared in the right lung a few years after operation and disappeared with x-ray therapy. Again 3 years ago he developed a right paramediastinal density which disappeared with x-ray therapy. Survived 13 years and 9 months, died of coronary thrombosis |
| Ochsner and DeCamp* | Pneumonectomy | There was never any clinical evidence of recurrence |
| Ochsner and DeCamp | Pneumonectomy | Well with no evidence of recurrence 5 years and 8 months after operation |
| Rose | Pneumonectomy | Died 4 years and 7 months after operation. Autopsy revealed no recurrence |
| Weissman and Christie | Pneumonectomy | Died 2 years after operation of generalized lymphosarcoma |

*The follow up information on these patients was obtained from the personal communications listed in the bibliography

TABLE I—CONT'D

| AUTHOR | AGE | SEX | X-RAY | SYMPTOMS | METASTASES | BRONCHOSCOPY AND CYTOLOGY |
|---|---|---|---|---|---|---|
| Sarokhan | 70 | M | Lingula | Chronic cough | None | Negative bronchial washings |
| Spatt and Grayzel | 34 | F | Right lower lobe, infiltrative mottled, egg-shaped area of density | Hemoptysis 3 months, cough | None | Suppuration, no cytology |
| Sugarbaker | No details—Primary in lung | | | | | |
| Touroff | — | — | Right upper lobe, 5 cm. | — | None | — |
| Dvoracek | 44 | M | Right upper lobe, massive | None | Parietal pleura, pericardium, peribronchial and hilar nodes | Compression, bronchial washings positive |
| Weissman and Christie | 52 | M | Borders poorly defined, left upper lobe, massive | Cough 1 year, DOE† 6 months, weight loss 6 lbs. | None | Negative, bronchial washings negative |
| Willis | 57 | M | Large opacity, right upper lobe | Recurrent pneumonia, 3 years | Hilar and peribronchial nodes | — |

All of the above patients were proved to have lymphosarcoma of the lung on pathologic examination of resected lung or at autopsy.

*Cited by other authors.

†Dyspnea of effort.

Microscopically, the tumor is composed of lymphocytes or lymphoblasts which replace the normal parenchyma. In the individual tumor the cells are of fairly uniform maturity. At the edges of the tumor the parenchyma is usually replaced in decreasing extent as it merges into the normal lung. As in 1 of our cases, there may be peripheral thin zones of fibrous tissue infiltrated by histiocytes. The neoplastic cells surround the vessels and bronchi but usually do not invade their walls.

No evidence of metastasis was found in 14 of the 23 cases. There was metastasis in all of the 5 autopsy and nonresectable cases. In 1 of the cases of resection, there was microscopic involvement of the peribronchial and hilar nodes. Recurrence in 4 of the patients who had resection has been mentioned.

## CASE REPORTS

CASE 1.—G. W., a 38-year-old white housewife, was admitted to the Barnes Hospital May 5, 1955, for investigation of an enlarging density found in chest films and an aching in the right shoulder and arm for four months. She had had chest films yearly for the past five years. One year prior to her admission, x-ray had shown a density in the right upper lobe. She had a work-up for tuberculosis which was said to show old arrested tuberculosis. One month previous to admission, a repeat film showed enlargement of the mass. She was bronchoscoped, had bronchograms made, and was told that she needed an operation. She denied hemoptysis, cough, sputum, and shortness of breath. Review of systems and past medical history was normal except for occasional intermittent attacks of epigastric and right upper quadrant cramping pain accompanied by nausea and vomiting and aggravated by fatty foods. There had been no weight loss

Physical examination was normal except for an area of consolidation over the anterior right upper lung field (Fig. 1A). Laboratory data including hemoglobin, white blood cell count, peripheral blood smear, urinalysis, serum Kahn test, and electrocardiogram were normal. Bronchoscopy revealed a slight granularity and edema at the right upper lobe orifice. Biopsy of the right upper lobe spur, bronchial washings, and three sputum examinations were negative for acid fast bacilli and tumor cells. Chest x rays revealed a large, rounded mass in the right upper lobe adjacent to the mediastinum, interpreted as questionable right upper lobe atelectasis, questionable mediastinal thymoma

On June 23, 1955, the patient was operated upon The anterior segment of the right upper lobe was a solid mass. Frozen section showed lymphosarcoma There were no palpable lymph nodes in the mediastinum or peribronchially The right upper lobe was removed in routine manner. Postoperatively the patient made an uneventful recovery and was discharged on the thirteenth postoperative day.

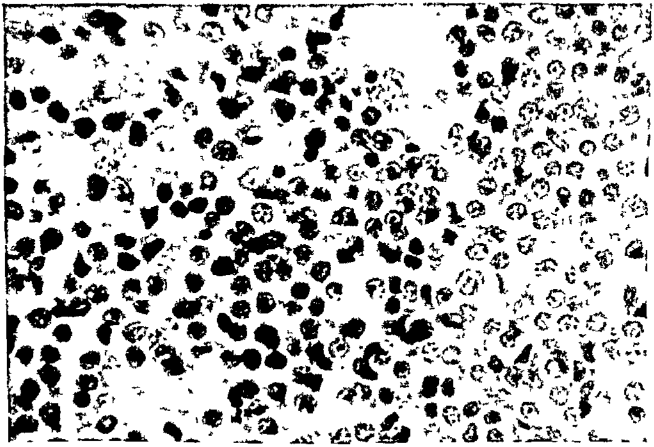


Fig 1C.—Case 1 Photomicrograph showing almost complete replacement of the normal parenchyma by sheets and lobulated or nodular aggregates of small relatively mature and fairly uniform lymphocytes

Grossly, there was a moderately firm tumor mass occupying the anterior segment of the right upper lobe extending into the adjacent apical and posterior segments (Fig. 1B). The margins of the tumor were distinct and accentuated by a 2 to 4 mm zone of firm gray yellow tissue. The cut surface of the tumor had a gray pink color with mottling of gray yellow, Microscopically, the excised rib showed normal marrow. Sections of the tumor showed almost complete replacement of the normal parenchyma by sheets and lobulated or nodular aggregates of small, relatively mature and fairly uniform lymphocytes (Fig 1C). Mitoses were seen occasionally Throughout the tumor were foci of patent alveoli with thickened walls invaded by lymphocytes At the periphery of the tumor was a zone of de
infiltrated by histiocytes The neoplastic cells completely surrounded but
walls of the arteries, veins, bronchial glands, nerves, and bronchi Seven h
contained no tumor.

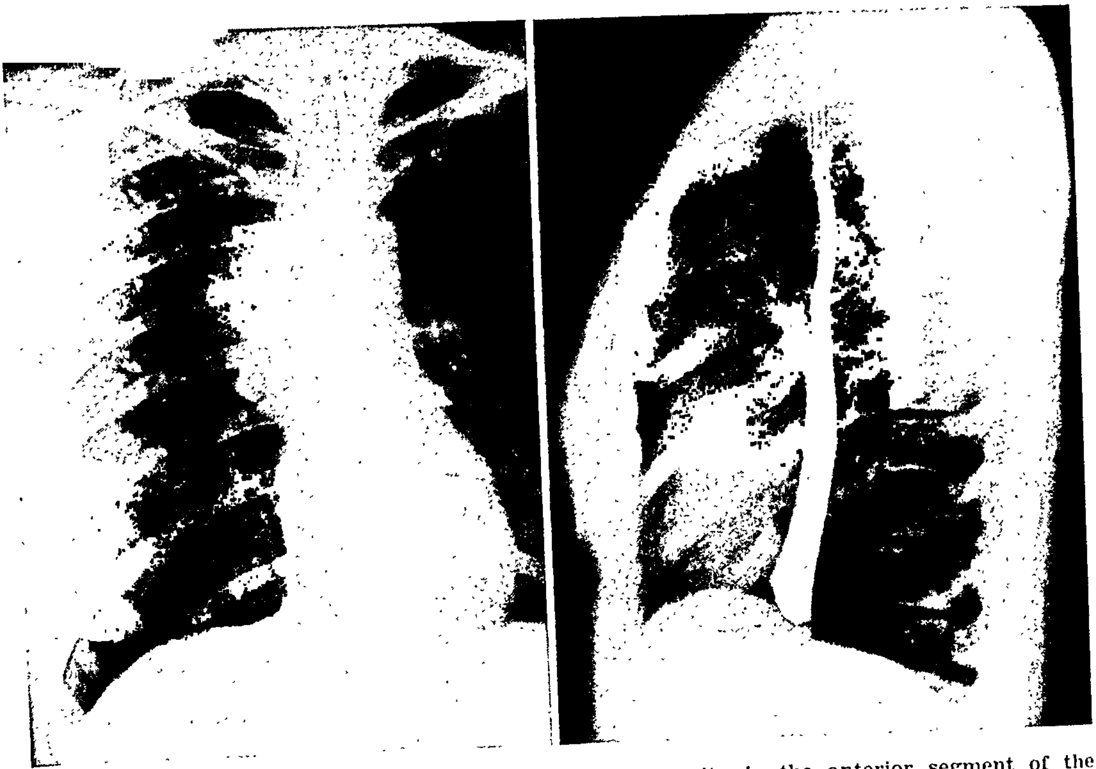


Fig. 1*A*.—Case 1. Admission films showing rounded density in the anterior segment of the right upper lobe.

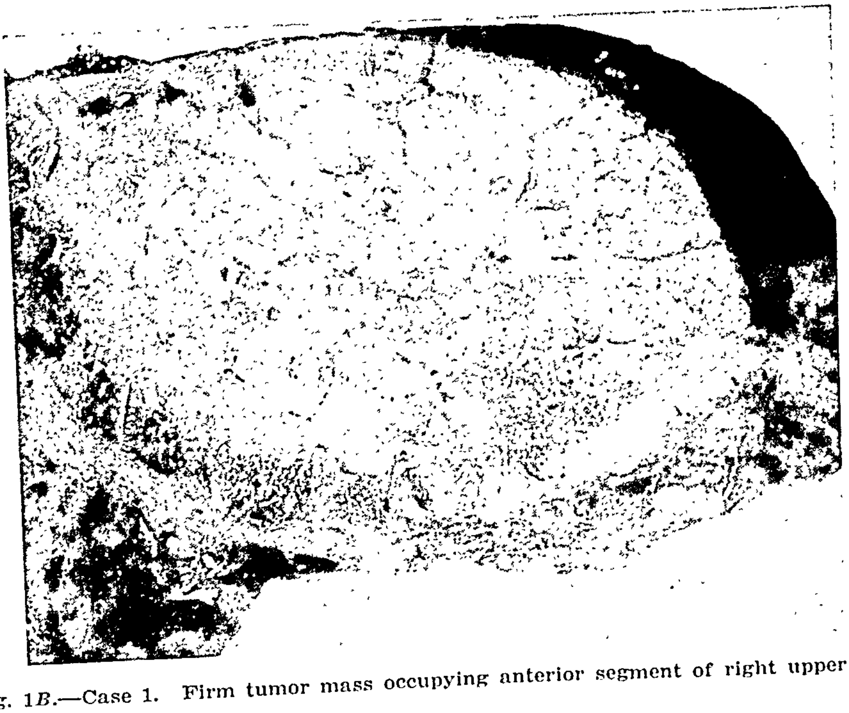


Fig. 1*B*.—Case 1. Firm tumor mass occupying anterior segment of right upper lobe.

The patient has been seen in follow-up, and there is no evidence of recurrence by symptoms or fluoroscopy six months after operation.

CASE 2.—N. L. P., a 75-year-old married white woman, was admitted to the Barnes Hospital on Dec. 19, 1948. In June, 1948, she had onset of left chest pain associated with an upper respiratory infection. Easy fatigability and malaise were the only other symptoms. In October she consulted her physician because of recurrent left anterior chest pain, not associated with effort. Fluoroscopy showed a shadow in the left upper lung field. Review of systems and past medical history was noncontributory except that eleven years previously she had had a thyroidectomy for mild symptoms of toxicity with resultant good regression of her symptoms. Physical examination revealed a well-nourished, mentally alert elderly white woman. Chest was clear to percussion and auscultation. There was no clubbing of the fingers. The remainder of the physical examination was essentially negative. Routine laboratory data including a red blood cell and white blood cell count, hemoglobin, urinalysis and peripheral blood smear were normal. Chest x-ray showed a wedge-shaped area of increased density in the left upper lobe (Fig. 2A). Bronchoscopy was performed on Dec. 21, 1948, with negative findings. Bronchial washings were taken and showed malignant cells. Three sputum examinations for acid-fast bacillus and for tumor cells were negative. Venous pressure and circulation time were normal. Gastric fill-up was normal. Preoperative diagnosis was bronchogenic carcinoma. On Dec. 27, 1948, a left thoracotomy was performed. The superior anteromedial portion of the left upper lobe revealed a reddish-gray lung tissue under which a very firm mass could be palpated. The hilus was free. Pneumonectomy was performed. The patient's postoperative course was entirely uneventful.

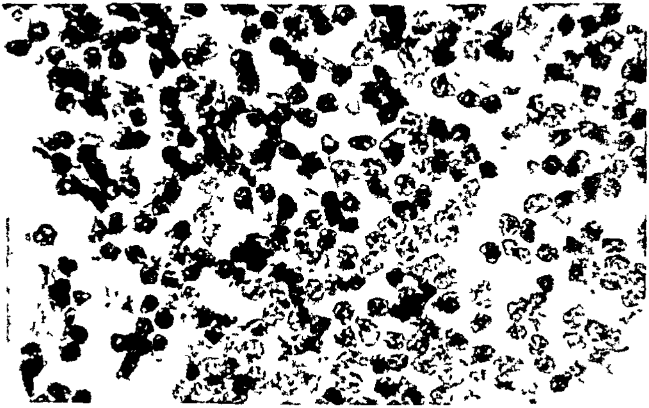


FIG. 2C.—Case 2. Photomicrograph showing replacement of the normal lung parenchyma by lymphocytes. Mitotic figures are frequent and there are a number of reticulum cells with prominent and multiple nucleoli.

Grossly, the specimen consisted of the entire left lung which weighed 395 Gm. In the upper lobe there was an atelectatic wedge-shaped area occupying the major portion of the apical segment of the upper lobe. There was a 6 cm. diameter, rounded, firm lesion palpable within the atelectatic segment. On cut section in the area of consolidation, there was a fleshy firm lesion approximately 6 to 7 cm. in greatest diameter (Fig. 2B). Grossly, the lung pattern appeared to be preserved and the lesion appear consist fleshy infiltration of the lung substance. There was no suggestion th within bronchi. The lesion was fairly well demarcated from t and of pale yellow color, but around the periphery there of

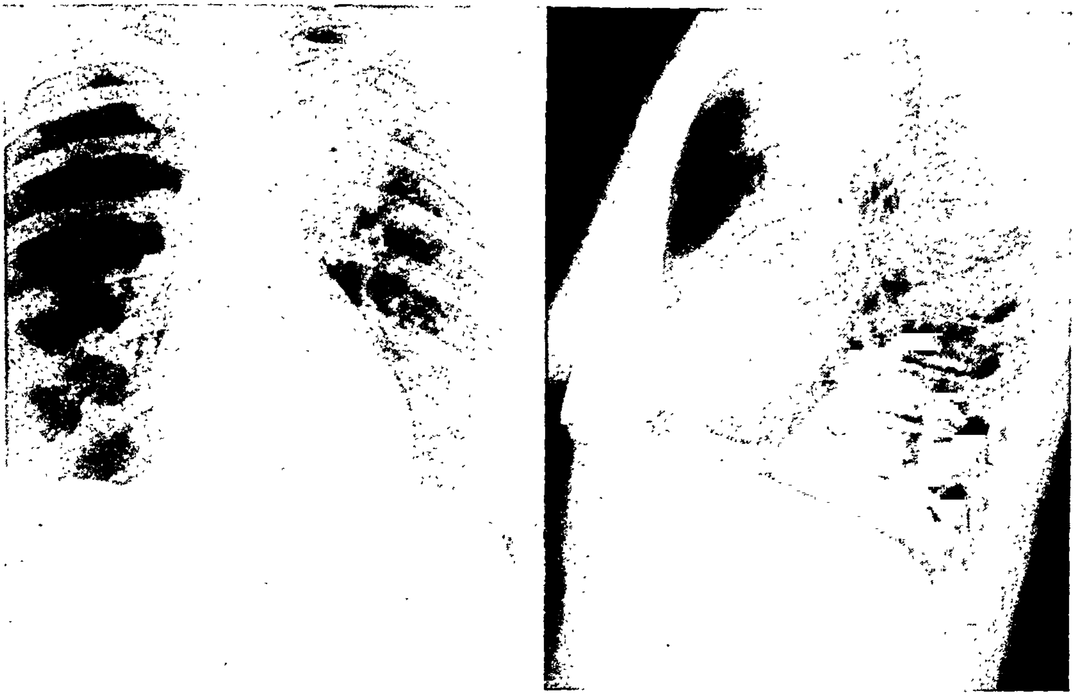
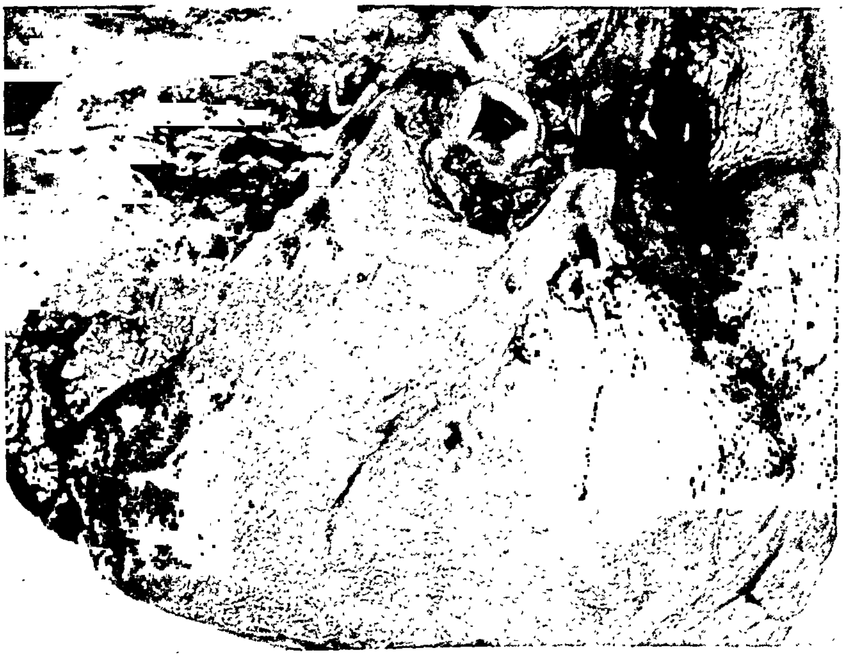


Fig. 2A.—Case 2. Admission films showing wedge-shaped area of increased density in the left upper lobe.



g. 2B.—Case 2. Wedge-shaped area of atelectasis containing the rounded fleshy tumor.

Dvoracek, C, and Cermak M  Primarni Lymfosarkom Plic, Lekarske Listy (Brno) 7 545, 1952
Grayzel, D M  Personal communication
Grimes, O F, Weirich, W, and Stephens, H B  Primary Lymphosarcoma of the Lung, J Thoracic Surg 27 378, 1954
Grimes, O F  Personal communication
Klassen, K P  Personal communication
Liebow, A A  Tumors of the Lower Respiratory Tract, Armed Forces Institute of Pathology, 1952, p 135
Pekelis, E  Linfosarcoma peribronchiale con sviluppo prevalentemente intrapolmonare rapido, Pathologica 23 66, 1931
Sirokhan, I, and Morrison, D A  Primary Lymphosarcoma of the Lung Ann Surg 138 140, 1953
Shallenberger, P L, Fisher, P, Beck, W C, and DeWan, C H  Concurrent Lymphoma of the Lung and Stomach, J Thoracic Surg 24 637, 1952
Spatt, S A, and Grayzel D M  Primary Lymphosarcoma of the Lung (A Case Report and Review of the Literature), Ann Int Med 27 632 1947
Sugarbaker, E D, and Craven, L F  Lymphosarcoma—A Study of 196 Cases With Biopsy, J A M A 115 17 23, 112 117, 1940
Touroff, A S W  Personal communication
Weissman, I, and Christie, J M  Primary Lymphosarcoma of the Lung (Report of a Case) Arch Surg 62 129, 1951
Wallis, R A  Pathology of Tumors, London, 1953, Butterworth & Co, Ltd

color shading off into the normal lung substance. Microscopically, the tumor had erased the normal pattern of the lung and had invaded the alveolar walls in other areas. Mitotic figures were seen rather frequently and there were a number of reticulum cells which had prominent and multiple nucleoli and were rarely multinucleated (Fig. 2*C*). No eosinophils or fibrosis was seen. Immediately adjacent to the main tumor in otherwise fairly normal lung tissue there were many areas of focal infiltration of the same type of cells seen in the main tumor mass. These were located in the subpleural, perivascular, and peribronchial regions. Parenchyma of the lung was otherwise normal. In one area, blood vessel invasion was seen and in one large bronchus the tumor was growing beneath the intact epithelium around the cartilage and out into the lung substance. Five lymph nodes from about the upper lung bronchus showed reticulum-cell hyperplasia and no tumor cells. Other nodes from the lower lobe bronchus and from the hilum were negative.

In June, 1953, four years and seven months after operation, the patient was admitted to another hospital in a moribund condition. She died shortly thereafter. Autopsy revealed intestinal obstruction due to adhesions and the examination showed no evidence of lymphosarcoma.

## DISCUSSION

Primary lymphosarcoma of the lung is distinctly different from the usual disseminated form of lymphosarcoma. It bears a similarity to lymphosarcoma of the stomach which often remains localized for long periods of time and may be cured by resection. Clinically, the lesion appears as an area of homogeneous increased density in the chest x-ray, which is not diagnostic. There are symptoms in less than one half of the reported cases. In only these instances has a diagnosis of malignancy been made preoperatively, one by bronchoscopic biopsy and two by cytologic sputum examination. At the time of operation the lesion may be recognized grossly as a neoplasm. Frozen section will identify its type.

There appears to be little difference in the recurrence rate between lobectomy and pneumonectomy. If the tumor is peripherally located and there are no grossly involved lymph nodes, lobectomy is recommended. Prognosis in resected cases is considerably better than for bronchogenic carcinoma.

Primary lymphosarcoma of the lung is another example of the need for exploratory thoracotomy in all cases in which there is a persistent unexplained esion in the chest x-ray.

## SUMMARY

Two patients with primary lymphosarcoma of the lung have been presented, with a summary of the literature. The need for exploratory thoracotomy in all cases in which there is an unexplained persistent lesion in the chest x-ray has been emphasized. The relatively good prognosis is apparent from this study.

## REFERENCES

Anlyan, A. J., Lovingood, C. B., and Klassen, K. P.: Primary Lymphosarcoma of the Lung, Surgery 27: 559, 1950.
Beck, W. C., and Reganis, J. C.: Primary Lymphoma of the Lung, J. THORACIC SURG. 22: 323, 1951.
Beck, W. C.: Personal communication.
Behrend, Moses: Personal communication.
Blalock, J. B.: Personal communication (for Ochsner and DeCamp).
Brown, C. J. O.: Personal communication.
Christie, J. M.: Personal communication.
Churchill, E. D.: Malignant Lymphoma of the Lung and Pulmonary Coccidiomycosis; Clinic on Surgical Lesions of the Lung, Surg. Clin. North America 27: 1113, 1947.
Churchill, E. D.: Personal communication.

Dvoracek C, and Cerval, M Primarni Lymfosarkom Plic Lekarske Listy (Brno) 7 545, 1952
Grivzel, D M Personal communication
Grimes, O F, Weirich, W and Stephens, H B Primary Lymphosarcoma of the Lung J Thoracic Surg 27 378 1954
Grimes, O F Personal communication
Klassen, K P Personal communication
Liebow, A A Tumors of the Lower Respiratory Tract, Armed Forces Institute of Pathology, 1952 p 135
Pelchis L Linfosarcoma peribronchiale con sviluppo prevalentemente intrapolmonare rapido, Pathologica 23 66, 1931
Sirokhan, I, and Morrison, D A Primary Lymphosarcoma of the Lung, Ann Surg 138 140, 1953
Shallenberger, P I, Fisher, P, Beck W C, and DeWan, C H Concurrent Lymphoma of the Lung and Stomach, J Thoracic Surg 24 637, 1952
Spratt, S A, and Grivzel D M Primary Lymphosarcoma of the Lung (A Case Report and Review of the Literature), Ann Int Med 27 632 1947
Sugarbaker, E D, and Craven L F Lymphosarcoma—A Study of 196 Cases With Biopsy, J A M A 115 17 23, 112 117, 1940
Touroff, A S W Personal communication
Weissman, I and Christie J M Primary Lymphosarcoma of the Lung (Report of a Case) Arch Surg 62 129 1951
Willis R A Pathology of Tumors, London 1953, Butterworth & Co, Ltd

color shading off into the normal lung substance. Microscopically, the tumor had erased the normal pattern of the lung and had invaded the alveolar walls in other areas. Mitotic figures were seen rather frequently and there were a number of reticulum cells which had prominent and multiple nucleoli and were rarely multinucleated (Fig. 2*C*). No eosinophils or fibrosis was seen. Immediately adjacent to the main tumor in otherwise fairly normal lung tissue there were many areas of focal infiltration of the same type of cells seen in the main tumor mass. These were located in the subpleural, perivascular, and peribronchial regions. Parenchyma of the lung was otherwise normal. In one area, blood vessel invasion was seen and in one large bronchus the tumor was growing beneath the intact epithelium around the cartilage and out into the lung substance. Five lymph nodes from about the upper lung bronchus showed reticulum-cell hyperplasia and no tumor cells. Other nodes from the lower lobe bronchus and from the hilum were negative.

In June, 1953, four years and seven months after operation, the patient was admitted to another hospital in a moribund condition. She died shortly thereafter. Autopsy revealed intestinal obstruction due to adhesions and the examination showed no evidence of lymphosarcoma.

## DISCUSSION

Primary lymphosarcoma of the lung is distinctly different from the usual disseminated form of lymphosarcoma. It bears a similarity to lymphosarcoma of the stomach which often remains localized for long periods of time and may be cured by resection. Clinically, the lesion appears as an area of homogeneous increased density in the chest x-ray, which is not diagnostic. There are symptoms in less than one half of the reported cases. In only these instances has a diagnosis of malignancy been made preoperatively, one by bronchoscopic biopsy and two by cytologic sputum examination. At the time of operation the lesion may be recognized grossly as a neoplasm. Frozen section will identify its type.

There appears to be little difference in the recurrence rate between lobectomy and pneumonectomy. If the tumor is peripherally located and there are no grossly involved lymph nodes, lobectomy is recommended. Prognosis in resected cases is considerably better than for bronchogenic carcinoma.

Primary lymphosarcoma of the lung is another example of the need for exploratory thoracotomy in all cases in which there is a persistent unexplained lesion in the chest x-ray.

## SUMMARY

Two patients with primary lymphosarcoma of the lung have been presented, with a summary of the literature. The need for exploratory thoracotomy in all cases in which there is an unexplained persistent lesion in the chest x-ray has been emphasized. The relatively good prognosis is apparent from this study.

## REFERENCES

Anlyan, A. J., Lovingood, C. B., and Klassen, K. P.: Primary Lymphosarcoma of the Lung, Surgery 27: 559, 1950.
Beck, W. C., and Reganis, J. C.: Primary Lymphoma of the Lung, J. THORACIC SURG. 22: 323, 1951.
Beck, W. C.: Personal communication.
Behrend, Moses: Personal communication.
Blalock, J. B.: Personal communication (for Ochsner and DeCamp).
Brown, C. J. O.: Personal communication.
Christie, J. M.: Personal communication.
Churchill, E. D.: Malignant Lymphoma of the Lung and Pulmonary Coccidiomycosis; Clinic on Surgical Lesions of the Lung, Surg. Clin. North America 27: 1113, 1947.
Churchill, E. D.: Personal communication.

made on the bronchial epithelium in routine (nontumor) necropsy specimens to obtain a base line and to set up criteria of normal, (2) those made on sections taken from the proximal portion of the bronchi in surgical specimens removed for conditions other than carcinoma, (3) those on the "normal appearing" proximal portion of the bronchus in surgical specimens removed at pneumonectomy for one of three types of carcinoma, namely, squamous cell carcinoma, adenocarcinoma, or small cell carcinoma, and (4) those made on necropsy specimens obtained from the good lung in cases in which carcinoma of the other lung had been treated surgically

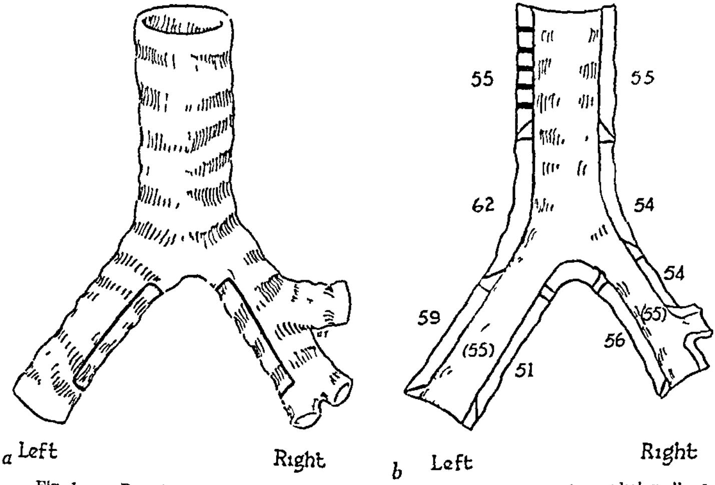


Fig 1—a Drawing to show where sections were taken from the posteromedial wall of the main stem bronchi of the routine necropsy specimens b Drawing to show where sections were taken from the lateral and medial walls of the trachea and primary bronchi of nonsmokers The numbers represent the average thickness in microns of the epithelium in the corresponding segments with the numbers in parentheses representing the over all average figures for the respective main stem bronchi

As demonstrated by Proetz[1] and later by Ermala and Holsti, smoke behaves according to certain predictable patterns The tars settle out at sites where eddy currents are set up by changes in direction of flow of the smoke or in cross sectional areas of the conducting system We felt that one such site, and one that is readily accessible for examination, is the medial and slightly posterior portion of the main stem bronchi consequently, we chose this site for study From the necropsy specimens a longitudinal strip approximately 3 to 5 cm in length was removed from the posteromedial wall of each bronchus (Fig 1 a) Longitudinal sections of these strips were made an stained with hematoxylin and eosin The slides were identified only by numb so that the observers had no knowledge about the individual represented by particular slide Of necessity, the strips of tissue removed from the surgical specimens were shorter than those obtained from the nec

# HISTOPATHOLOGIC OBSERVATIONS ON BRONCHIAL EPITHELIUM WITH SPECIAL REFERENCE TO CARCINOMA OF THE LUNG

Robert F. Ryan, M.D.,* John R. McDonald, M.D.,** and
O. Theron Clagett, M.D.***
Rochester, Minn.

THE possibility that smoking is a cause of carcinoma of the lung is being intensively and widely discussed at present. In consideration of this question, the histologic changes produced in the lung by smoking are of fundamental interest, and for this reason we are presenting the results of a study that we have made of such changes in man.

When we first started this study, we planned to compare histologic sections from the lungs of smokers with sections from the lungs of nonsmokers, in a manner analogous to the procedure we have used to study the laryngeal epithelium of smokers and nonsmokers.[1, 2] Superficially, this approach of studying histologic sections from the lungs might seem to be an easy one, but in actual practice there are at least three major difficulties: First, there seem to be no answers to such questions as: What is the normal appearance of the bronchial epithelium? How thick should it be? What effect has age on the epithelium? Is there a difference between the bronchial epithelium of males and that of females? Is there a difference between the right and left sides? Is squamous metaplasia normal? How often does it occur in normal people? Is it more frequent with advancing age? These are but a few of the basic questions that have to be answered before a specimen of epithelium may be designated as abnormal or normal. The second major difficulty is that other conditions, such as chronic bronchitis, inhalation of industrial fumes, pulmonary edema, and terminal coma, affect the appearance of the bronchial epithelium. The third major difficulty is that of obtaining histories of smoking or nonsmoking that are reliable. Moreover, even after obtaining reliable histories, one has to be very careful to ascertain that the smokers are representative of *all* smokers and that some factor of selection has not entered into the study, as pointed out by Berkson.[3]

### MATERIAL AND PROCEDURE

Because of the difficulties mentioned, we found it advisable to approach this problem mainly from a pathologic standpoint. We used four categories of specimens, and hence our observations are grouped as follows: (1) those

---

The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

Received for publication Jan. 20, 1956.

*Fellow in Surgery, Mayo Foundation, Rochester, Minn.

**Section of Surgical Pathology, Mayo Clinic and Mayo Foundation, Rochester, Minn.

***Section of Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minn.

we were careful, in removing the strips beginning at the proximal end of the bronchi in the surgical specimens and extending distally toward the tumor, to stop well short of the visible margins of the tumor, the reason for this care is the fact that, as pointed out by Habein,[6] a zone of carcinoma in situ often extends out from the tumor but remains in continuity with the primary lesion By the use of a stencil, ink marks were placed on each slide to designate the areas where the epithelial thickness was to be measured Measurements were made only in those areas where no erosions or artifacts were present, for example, we have observations on the incidence of squamous metaplasia occurring in the bronchus of 30 left lungs removed because of squamous cell carcinoma, but determinations of epithelial thickness were made on only 25 of these lungs

In the previous studies of the effect of smoking on the larynx[1, 3] there were 5 persons who, it seemed certain, were nonsmokers and from whom bronchial tissue was preserved In 2 of these cases a strip of tissue was removed from each main stem bronchus, as was done in the routine necropsy cases mentioned above, but in the other 3 cases, additional strips were removed from the lateral walls of the distal part of the trachea and the proximal portions of each bronchus (Fig 1, *b*)

### RESULTS

*Routine Necropsy Specimens*—In the routine (nontumor) necropsy specimens, squamous metaplasia was not frequently encountered It was present in 6 of the 77 males (7 8 per cent), or in 4 of 65 left bronchi and in 5 of 61 right bronchi It was found in 2 of the 36 females (5 6 per cent), or in one of 28 left bronchi and in 2 of 29 right bronchi No evidence of malignancy was seen in any of the routine necropsy specimens, either from males or from females The data on thickness of the bronchial epithelium are given in Table I* It is to be noted that the bronchial epithelium from the routine necropsy specimens was thicker in the males than in the females, and that while the bronchial epithelium on the right was of the same thickness as that on the left in the females it was thicker than that on the left in the males These subjects were distributed through the second to tenth decades of life inclusive, with the majority being rather evenly distributed through the fifth to ninth decades We found no consistent relationship between age and epithelial thickness, or between age and the occurrence of squamous metaplasia in these routine necropsy specimens

In Table I, under the heading "Necropsy specimens without carcinoma of lung" and under the subheading "Normal," 10 specimens (5 cases) are listed All 5 patients were males Two of the 5 were farmers, one was a barber, one was a carpenter and one had been an inmate of a state mental hospital for many years The carpenter lived in a town of less than 25,000 population and the others were essentially rural residents, living in towns of 2,000 or 3,000 population Thus, urban living, in its usual sense was ruled

---

*Detailed tables on epithelial thickness of specimens in all four categories a according to distribution of patients by age will be supplied by the authors on

TABLE I. THICKNESS OF EPITHELIUM IN 387 BRONCHI (294 SUBJECTS)

| | NECROPSY SPECIMENS WITHOUT CARCINOMA OF LUNG | | | | SURGICAL SPECIMENS | | | | | | NECROPSY SPECIMENS WITH CARCINOMA OF LUNG | | | | TOTAL | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NORMAL* | | ROUTINE | | SQUAMOUS-CELL CARCINOMA | | ADENOCARCINOMA (AND ADENOMA) | | SMALL-CELL CARCINOMA | | OPPOSITE LUNG | | LUNG WITH TUMOR (BRONCHIAL STUMP) | | | |
| | NO. | MEAN† | NO. | MEAN† | NO. | MEAN† | NO. | MEAN† | NO. | MEAN† | NO. | MEAN† | NO. | MEAN† | NO. | MEAN† |
| *Males* | | | | | | | | | | | | | | | | |
| Left | 5 | 55 | 65 | 64.6 | 25 | 72.6 | 13 | 66.8 | 21 | 70.3 | 21 | 79.4 | 5 | 77.0 | 155 | 67.3 |
| Right | 5 | 55 | 61 | 70.2 | 23 | 64.0 | 25 | 69.1 | 16 | 71.5 | 15 | 73.1 | 11 | 80.9 | 156 | 68.2 |
| *Females* | | | | | | | | | | | | | | | | |
| Left | | | 28 | 60.8 | | | 9 | 67.6 | 1 | 75 | 3 | 66.7 | 1 | 60 | 42 | 63.4 |
| Right | | | 29 | 60.5 | | | 2 | 70 | | | 2 | 52.5 | 1 | 65 | 34 | 60.7 |
| Total | 10 | 55 | 183 | 65.2 | 48 | 68.4 | 49 | 68.2 | 38 | 70.9 | 41 | 74.9 | 18 | 77.8 | 387 | 66.7 |

*Normal as defined in the text.

†Mean epithelial thickness in microns.

basilar hyperplasia to frank carcinoma such as has been described in the uterine cervix can be seen in our sections from these opposite or "good" lungs. Fig. 6 portrays sections from a 63-year-old white man who underwent right pneumonectomy for a squamous-cell carcinoma in 1949. In 1952, the patient was found to be well and presented no evidence of any recurrence of his disease. While he was eating in a downtown restaurant, he aspirated a bolus of meat and died of asphyxia. At necropsy the left bronchus appeared normal on gross examination, but the photomicrographs show that pathologic changes were present; the patient did not live long enough for the lesions to become symptomatic.

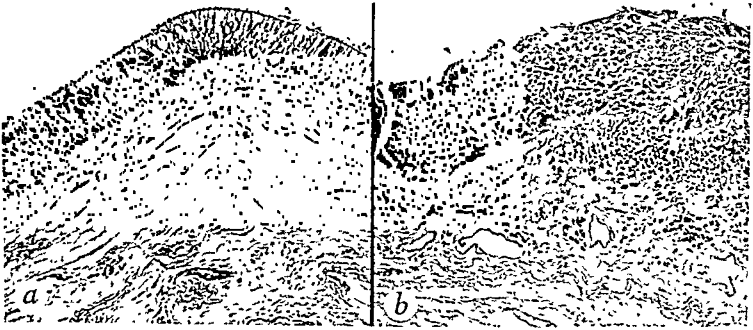


Fig. 6.—*a*, Marked hyperplasia of the surface epithelium of the left main-stem bronchus in a 63-year-old man who had undergone right pneumonectomy for squamous-cell carcinoma some three years previously (see text for details). In 1952, while he was in town for a checkup, he died of asphyxia from a bolus of meat. Post-mortem examination disclosed no gross evidence of residual carcinoma (hematoxylin and eosin; ×100). *b*, Photomicrograph of bronchial epithelium from the left lung of the same subject as in *a*. This area adjacent to the tissue shown in *a* shows marked squamous metaplasia with an in situ carcinoma of the surface epithelium.

## COMMENT

At the time this study was undertaken, almost two years ago, we were not familiar with any similar attempts to evaluate the effects of tobacco on the lungs. Subsequently, we found three other studies of a somewhat similar nature. The first is the report of Wittekind and Strüder,[7] who studied surgical and necropsy specimens to see if smoking produced any effect on the bronchi. These authors concluded that no relationship existed between tobacco smoking and the bronchial structure. They felt that squamous metaplasia and any other pathologic changes present were due to chronic bronchitis. They did find a high percentage of smokers among those having carcinoma of the lung. In 4 of their cases there were small, in situ cancers in addition to the larger and obvious tumor of the lung. The second study is that of Weller,[8, 9] in which 21 sections were taken from the lungs in a set pattern at necropsy. The sections of epithelium were studied microscopically for squamous metaplasia, both typical and atypical, and the findings were then correlated with age, sex, occupation, smoking habits, and place of residence. With 21 sections, Weller found a much higher incidence of squamous metaplasia than we have observed

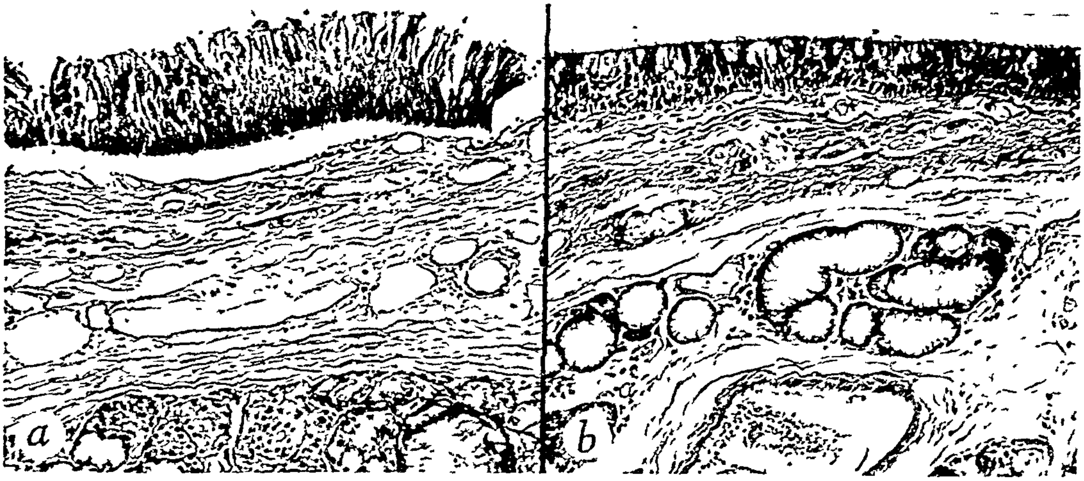


Fig. 4.—*a*, Marked basilar hyperplasia of the bronchial epithelium of the left main-stem bronchus. The patient had a squamous-cell carcinoma of the right lung (hematoxylin and eosin; ×100). *b*, Moderate basilar hyperplasia of the bronchial epithelium. This specimen is from the right lung of a 60-year-old man who had a squamous-cell carcinoma of the left lung (hematoxylin and eosin; ×100).

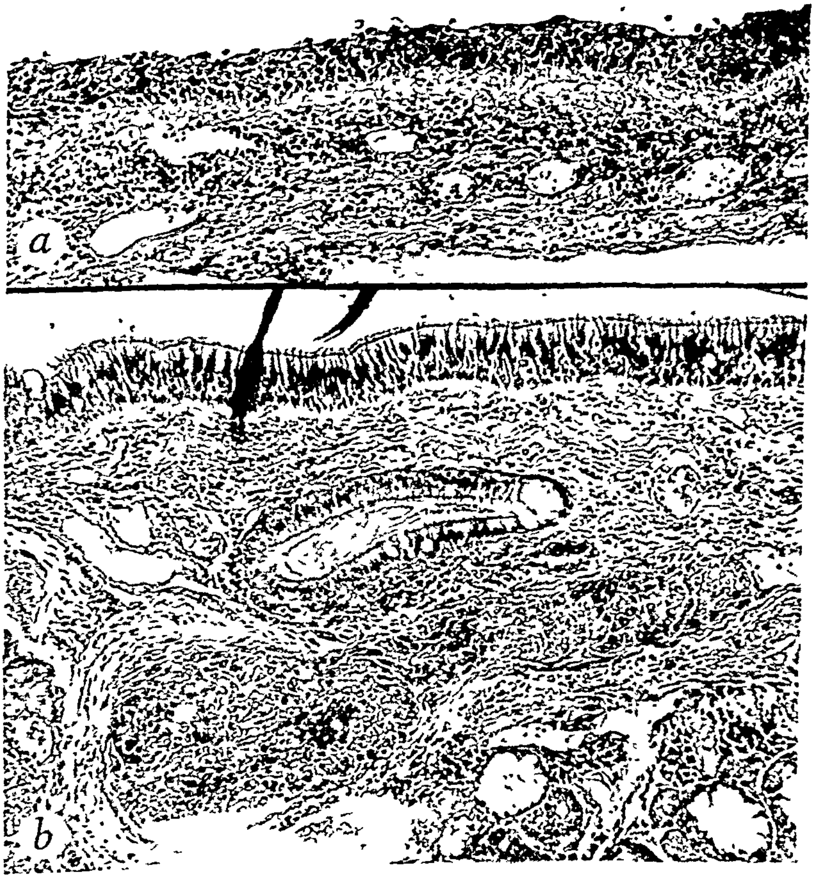


Fig. 5.—*a*, Hyperpla [illegible] metaplasia [illegible] bronchial epithelium of the left
g from a 62-year-old [illegible] amous [illegible] of the right lung (hema-
ylin and eosin; ×100). [illegible] the su [illegible] m and an in situ carcinoma
a bronchial gland. This [illegible] 65-year-old man who had
*quamous-cell carcinoma* [illegible] ×100).

It is interesting that the epithelium of the right main stem bronchus was thicker than that of the left in the males of the routine necropsy series. Although the increase in thickness in the cases of carcinoma was not as great as in the routine necropsy series, the average figures for the right side were more than those of the left. It is also to be noted that the over all incidence of squamous metaplasia was greater on the right than on the left side, with more occurring in males than in females. It was our feeling that in most cases squamous metaplasia seemed to start at or near the opening of ducts and occasionally in the bronchial glands.

In our previously reported study of the larynx we noted that squamous metaplasia was present in the false cord or subglottic area in about one half of the male larynxes and in one fourth of the female larynxes. It is certainly an attractive speculation that some agent in the inspired air is one of the main causes of squamous metaplasia of the respiratory tract, with a high incidence of such lesions in the larynx, where concentration of the agent would be very high, and a much lower incidence in the main stem bronchi where considerable dilution has taken place. One would not expect such an agent to have much effect on most of the trachea, as this is a relatively straight tube in which no eddy currents or areas of turbulence should be present to cause the agents, such as tars in tobacco smoke, to be deposited on the walls. The work of Ermala and Holsti is interesting in this light. They made a plastic model of the respiratory tract and, coating it with egg albumen made it smoke, this caused the inspired tars to be deposited in the larynx and main-stem bronchi as shown in Fig. 7, *a*. Fig. 7, *b* shows the areas where carcinoma occurs in the respiratory tract. These authors state that there is a "striking correlation noted between the clinical frequency of cancer and the localization of the tobacco smoke, especially in view of smoking habits."

Another observation of great interest in our study is the relatively high incidence of in situ lesions or small carcinomas in the opposite (good) lung in cases of carcinoma of the lung. Is carcinoma of the lung a multicentric disease? These patients did not live long enough for the lesions in the opposite lung to assume clinical significance. The question comes up, How long does it take pulmonary lesions to develop? Does it take years, as in the case of in situ lesions of the uterine cervix?

Finally, this study raises the question of how much of the bronchial epithelium proximal to a tumor should be removed. This is most important in the cases of squamous cell carcinoma and small cell carcinoma, in which a high incidence of atypical squamous metaplasia was seen in this study. Changes of this nature seem to be one more indication for pneumonectomy rather than lobectomy in these cases if such is at all feasible.

## SUMMARY AND CONCLUSIONS

This study shows that in the absence of irritants such as industrial fumes and smoking the normal bronchial epithelium of males is a pseudostratified columnar epithelium about 55 $\mu$ thick. In routine necropsy cases the thickness of the epithelium is about 67 $\mu$ in males and 60 $\mu$ in fema[illegible] in our routine necropsy cases, in which only one block was removed from each main-stem bronchus. In neither of the two reports do the authors give any data on thickness of the bronchial epithelium in microns. The third report is that of Auerbach and associates,[10] who found that smokers had thicker bronchial epithelium and also a higher incidence of metaplasia than did nonsmokers.

In our study, we found that the bronchial epithelium is thicker in the male than in the female, which corresponds to the findings in our studies on the laryngeal epithelium. We found no consistent relationship between thickness of the bronchial epithelium and age.

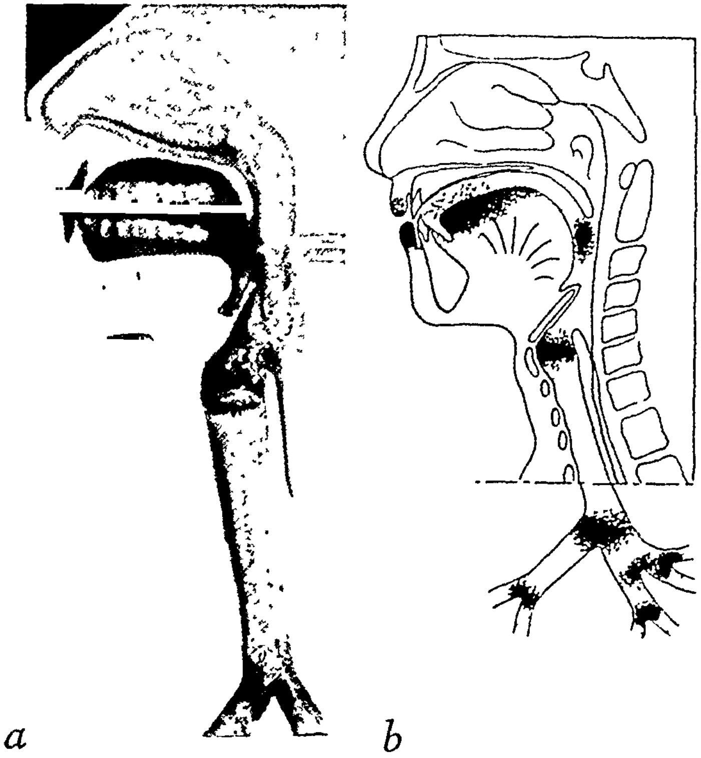


Fig. 7.—*a*, Plastic model of the respiratory tract showing the deposition of tobacco tars. *b*, Sketch showing the sites of carcinoma of the respiratory organs (From Ermala, and Holsti: Cancer 8: 674-678, 1955.)

In the routine necropsy cases, the finding of squamous metaplasia in 7.8 per cent of the cases is in sharp contrast with the finding in the surgical specimens from cases of carcinoma of the lung, in which 78 per cent of the squamous-cell carcinomas (all males), 34 per cent of the adenocarcinomas, and 71 per cent of the small-cell carcinomas were associated with squamous metaplasia of the bronchial epithelium proximal to the tumors. Much of this might be explained on the basis of irritation and secondary infection due to the tumors, but this basis is inadequate to explain the 44 per cent incidence of squamous metaplasia or the 12 per cent incidence of in situ or frank carcinoma in what appeared grossly to be normal surface epithelium in the opposite (good) lung.

# RESISTANCE TO AIR FLOW IN BRONCHOSPIROMETRIC CATHETERS

E W Swenson, M D, and G Birath, M D

With the Technical Assistance of A Åhbeck

GAENSLER and his associates[1] have studied certain physical principles bearing on breathing against the resistance due to the turbulent type of flow ordinarily encountered in bronchospirometric catheters and have shown that any decrease in airway diameter results in a marked increase in resistance. The various catheters tested by these authors exhibited from four to eighteen times the resistance of the normal human airway. The Carlens tube[2] was found to be superior in this and other respects to the Norris,[3] Zavod,[4] and Gebauer[5] catheters.

The Marsh catheter[6] was introduced subsequent to the study just mentioned[1] and, in addition to certain mechanical improvements, was said to have less resistance to breathing than others; however, no data were given to support this statement. Since bronchospirometry has been utilized extensively in this laboratory, it was decided to investigate and compare the air flow resistance characteristics of the various catheters including that of Marsh, using both moist and dry air; moist air is usually respired during bronchospirometry and the influence of its increased density was not investigated by Gaensler and his co-workers.

## METHODS

The apparatus used was similar to that of Gaensler, and is shown in Fig. 1. The only major modification was an effort to saturate the air flowing from the tank with water vapor. Studies were also made for comparison using dry air.

The flow rate, controlled at the valve of a compressed air tank, was varied between zero and 50 L. per minute and measured with a calibrated flow meter.* The maximum figure was derived from the highest minute ventilation value observed in our series of over 500 bronchospirometries, and from multiplying this value by 3.3 to estimate maximum rate of flow occurring during the respiratory cycle.[7] Pressures existing in the system were measured at the various flow rates without catheter and with each lumen of the following catheters:

| CATHETER | EXTERNAL DIAMETER (MM.) | INTERNAL CROSS SECTIONAL AREA (CM.²) | |
|---|---|---|---|
| | | RIGHT | LEFT |
| Carlens 41 Fr | 15 | 0.44 | 0.45 |
| Carlens 39 Fr | 13 | 0.38 | 0.36 |
| Carlens 37 Fr | 12.5 | 0.31 | 0.33 |
| Marsh | 14 | 0.28 | 0.27 |
| Zavod | 12 | 0.20 | 0.20 |

---

From the Pulmonary Function Laboratory, Renströmska Sjukhuset, Gothenburg, Sweden.
Received for publication Jan. 27, 1956.

*Model G 9148B, Emil Greiner Co., New York 13, New York.

thelium in the male is almost always thicker than that of the female, and in the males the epithelium of the right bronchus tends to be thicker than that of the left. The incidence of squamous metaplasia was approximately 8 per cent in the routine necropsy cases of this study. The epithelium proximal to a carcinoma and the epithelium in the opposite (good) lung tend to be thicker and more hyperplastic and to present a much higher incidence of squamous metaplasia, much of which is atypical, than does the normal bronchial epithelium. A 12 per cent incidence of in situ or frank carcinoma was found on microscopic examination of the opposite ("good") lung in cases of carcinoma of the lung even though the bronchi appeared normal on gross examination.

## REFERENCES

1. Ryan, R. F., McDonald, J. R., and Devine, K. D.: The Pathologic Effects of Smoking on the Larynx, A. M. A. Arch. Path. 60: 472-480, 1955.
2. Ryan, R. F., McDonald, J. R., and Devine, K. D.: Changes in Laryngeal Epithelium: Relation to Age, Sex and Certain Other Factors, Proc. Staff Meet., Mayo Clin. 31: 47-52, 1956.
3. Berkson, J.: The Statistical Study of Association Between Smoking and Lung Cancer, Proc. Staff Meet., Mayo Clin. 30: 319-348, 1955.
4. Proetz, A.: Some Preliminary Experiments in the Study of Cigarette Smoke and Its Effects Upon the Respiratory Tract, Ann. Otol., Rhin. & Laryng. 48: 176-194, 1939.
5. Ermala, P., and Holsti, L. R.: Distribution and Absorption of Tobacco Tar in the Organs of the Respiratory Tract, Cancer 8: 673-678, 1955.
6. Habein, H. C., Jr.: Recurrent Carcinoma in the Bronchial Stump. Thesis, Graduate School, University of Minnesota, 1954.
7. Wittekind, D., and Strüder, R.: Beitrag zur Histogenese des Bronchialcarcinoms. I. Über Epithelmetaplasie im Bronchialbaum. II. Über die Beziehungen zwischen Epithelmetaplasien und Carcinombildung im Bronchialbaum, Frankfurt. Ztschr. Path. 64: 294-311; 405-437, 1953.
8. Weller, R. W.: Metaplasia of Bronchial Epithelium: A Postmortem Study, Am. J. Clin. Path. 23: 768-774, 1953.
9. Weller, R. W.: Bronchial Mucosa: Relation to Occupation, Residence, and Smoking. Read at the meeting of the American Medical Association, Atlantic City, N. J., June 6-10, 1955.
0. Auerbach, O.: Personal communication to the authors.

# RESISTANCE TO AIR FLOW IN BRONCHOSPIROMETRIC CATHETERS

E. W. Swenson, M.D., and G. Birath, M.D.

With the Technical Assistance of A. Åhbeck

GAENSLER and his associates[1] have studied certain physical principles bearing on breathing against the resistance due to the turbulent type of flow ordinarily encountered in bronchospirometric catheters, and have shown that any decrease in airway diameter results in a marked increase in resistance. The various catheters tested by these authors exhibited from four to eighteen times the resistance of the normal human airway. The Carlens tube[2] was found to be superior in this and other respects to the Norris,[3] Zavod,[4] and Gebauer[5] catheters.

The Marsh catheter[6] was introduced subsequent to the study just mentioned[1] and, in addition to certain mechanical improvements, was said to have less resistance to breathing than others; however, no data were given to support this statement. Since bronchospirometry has been utilized extensively in this laboratory, it was decided to investigate and compare the airflow resistance characteristics of the various catheters, including that of Marsh, using both moist and dry air; moist air is usually respired during bronchospirometry and the influence of its increased density was not investigated by Gaensler and his co-workers.

## METHODS

The apparatus used was similar to that of Gaensler, and is shown in Fig. 1. The only major modification was an effort to saturate the air flowing from the tank with water vapor. Studies were also made for comparison using dry air.

The flow rate, controlled at the valve of a compressed air tank, was varied between zero and 50 L. per minute and measured with a calibrated flow meter.* The maximum figure was derived from the highest minute ventilation value observed in our series of over 500 bronchospirometries, and from multiplying this value by 3.3 to estimate maximum rate of flow occurring during the respiratory cycle.[7] Pressures existing in the system were measured at the various flow rates without catheter and with each lumen of the following catheters:

| CATHETER | EXTERNAL DIAMETER (MM.) | INTERNAL CROSS-SECTIONAL AREA (CM.²) | |
|---|---|---|---|
| | | RIGHT | LEFT |
| Carlens 41 Fr. | 15 | 0.44 | 0.45 |
| Carlens 39 Fr. | 13 | 0.38 | 0.36 |
| Carlens 37 Fr. | 12.5 | 0.31 | 0.33 |
| Marsh | 14 | 0.28 | 0.27 |
| Zavod | 12 | 0.20 | 0.20 |

---

From the Pulmonary Function Laboratory, Renströmska Sjukhuset, Gothenburg, Sweden.
Received for publication Jan. 27, 1956.

*Model G 9148B, Emil Greiner Co., New York 13, New York thelium in the male is almost always thicker than that of the female, and in the males the epithelium of the right bronchus tends to be thicker than that of the left. The incidence of squamous metaplasia was approximately 8 per cent in the routine necropsy cases of this study. The epithelium proximal to a carcinoma and the epithelium in the opposite (good) lung tend to be thicker and more hyperplastic and to present a much higher incidence of squamous metaplasia, much of which is atypical, than does the normal bronchial epithelium. A 12 per cent incidence of in situ or frank carcinoma was found on microscopic examination of the opposite ("good") lung in cases of carcinoma of the lung even though the bronchi appeared normal on gross examination.

## REFERENCES

1. Ryan, R. F., McDonald, J. R., and Devine, K. D.: The Pathologic Effects of Smoking on the Larynx, A. M. A. Arch. Path. 60: 472-480, 1955.
2. Ryan, R. F., McDonald, J. R., and Devine, K. D.: Changes in Laryngeal Epithelium: Relation to Age, Sex and Certain Other Factors, Proc. Staff Meet., Mayo Clin. 31: 47-52, 1956.
3. Berkson, J.: The Statistical Study of Association Between Smoking and Lung Cancer, Proc. Staff Meet., Mayo Clin. 30: 319-348, 1955.
4. Proetz, A.: Some Preliminary Experiments in the Study of Cigarette Smoke and Its Effects Upon the Respiratory Tract, Ann. Otol., Rhin. & Laryng. 48: 176-194, 1939.
5. Ermala, P., and Holsti, L. R.: Distribution and Absorption of Tobacco Tar in the Organs of the Respiratory Tract, Cancer 8: 673-678, 1955.
6. Habein, H. C., Jr.: Recurrent Carcinoma in the Bronchial Stump. Thesis, Graduate School, University of Minnesota, 1954.
7. Wittekind, D., and Strüder, R.: Beitrag zur Histogenese des Bronchialcarcinoms. I. Über Epithelmetaplasie im Bronchialbaum. II. Über die Beziehungen zwischen Epithelmetaplasien und Carcinombildung im Bronchialbaum, Frankfurt. Ztschr. Path. 64: 294-311; 405-437, 1953.
8. Weller, R. W.: Metaplasia of Bronchial Epithelium: A Postmortem Study, Am. J. Clin. Path. 23: 768-774, 1953.
9. Weller, R. W.: Bronchial Mucosa: Relation to Occupation, Residence, and Smoking. Read at the meeting of the American Medical Association, Atlantic City, N. J., June 6-10, 1955.
10. Auerbach, O.: Personal communication to the authors.

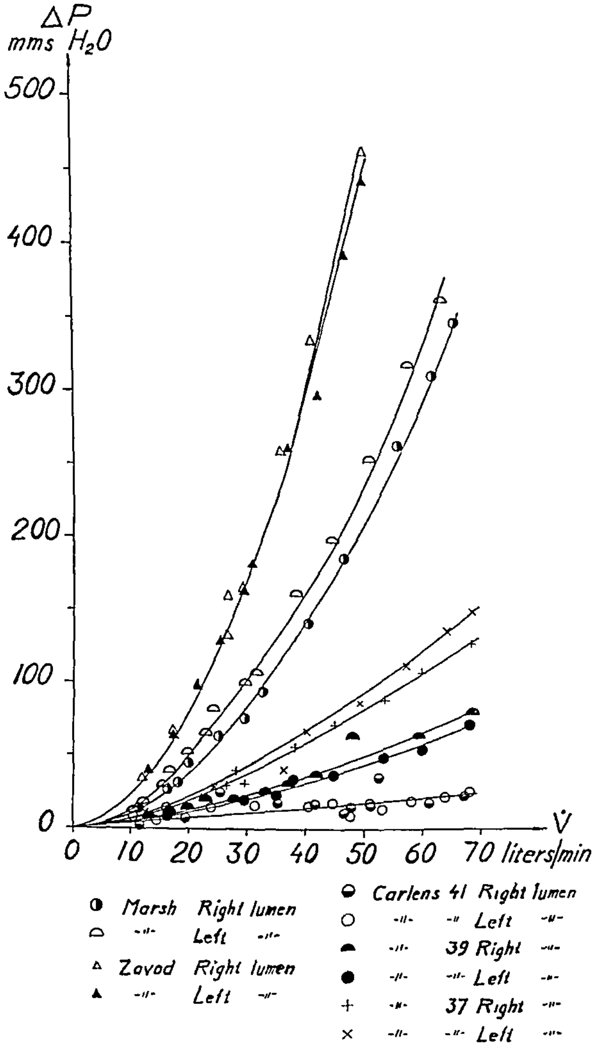


Fig. 2.—Flow rate-resistance relationships in various bronchospirometric catheters. Dry air. The tubes with smaller diameters develop high resistance (ΔP) as the flow rate ($\dot{V}$) is increased. The curves of the high resistance tubes indicate turbulent flow whereas the larger catheters allow streamline flow.

Pressures were recorded in each case as $\Delta$ P in centimeters $H_2O$, the difference between the observed pressure within the system with the catheter in place and the pressure found at the same flow rate when no catheter was present.

## RESULTS

Fig. 2 graphically portrays the back pressures exerted due to resistance in each catheter lumen at the various flow rates for *dry* gas. In Fig. 3 the same data are presented with the pressures plotted on a square scale; the relationship between flow and pressure becomes almost rectilinear except for the Carlens 41 catheter (see discussion) and the first part of the curves during slow rates of flow. Each of the two lumina of the Marsh catheter have somewhat more resistance than do those of any of the Carlens tubes, and somewhat less than those of the Zavod catheter.

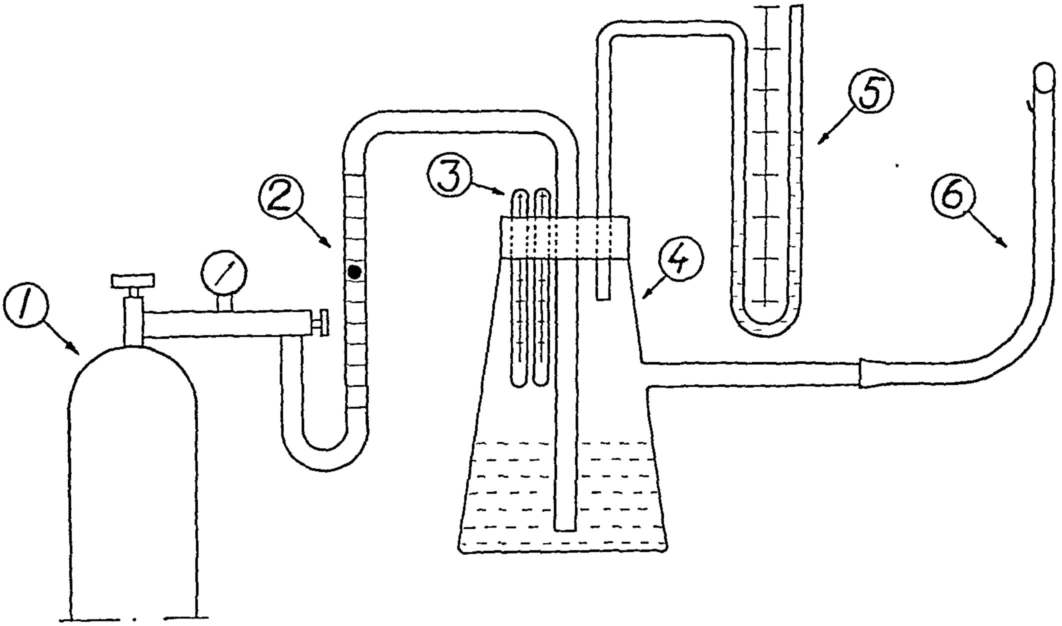


Fig. 1.—Arrangement of apparatus to measure pressures due to resistance to air flow in bronchospirometric catheters. Air flow was controlled at the compressed air tank (*1*) and measured at the flowmeter (*2*). Saturation of the air with water vapor took place when the air bubbled through water in a large flask (*4*) and was measured with a psychrometer (*3*). For each flow rate, back pressure, *P*, due to the catheter (*6*) was read at the manometer (*5*).

Similar relationships between the various catheters are noted when moist air is used instead of dry. During this second series of experiments, psychrometer readings ranged from 92 to 99 per cent relative humidity at the various flow rates. All pressures are relatively higher than those found when dry air is used (Fig. 4).

## DISCUSSION

The back pressure, $\Delta$ P, resulting from a given nonturbulent, uniform flow through a smooth, straight tube is predictable in accordance with

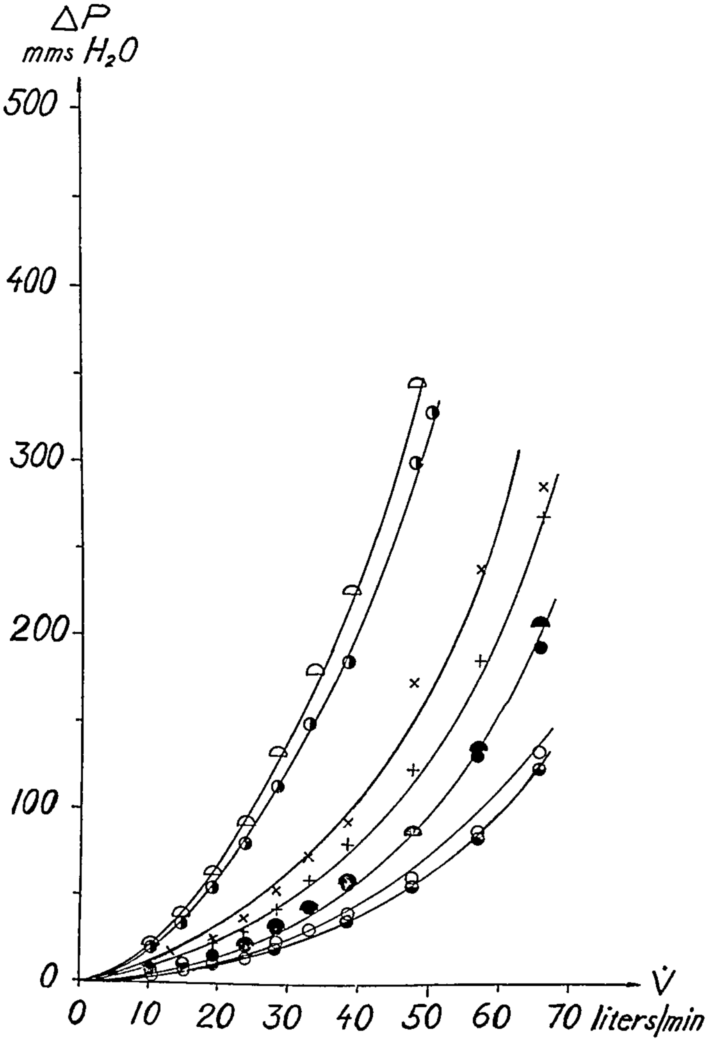


FIG. 4—Flow rate-resistance relationships in the same bronchospirometeric catheters. Moist air. The resistance of each tube is again seen to be related to its internal cross-sectional dimensions. However, the addition of water vapor (simulating conditions of clinical use) results in turbulent flow at all flow rates and consequently a sharply increased resistance even in the large lumen catheters.

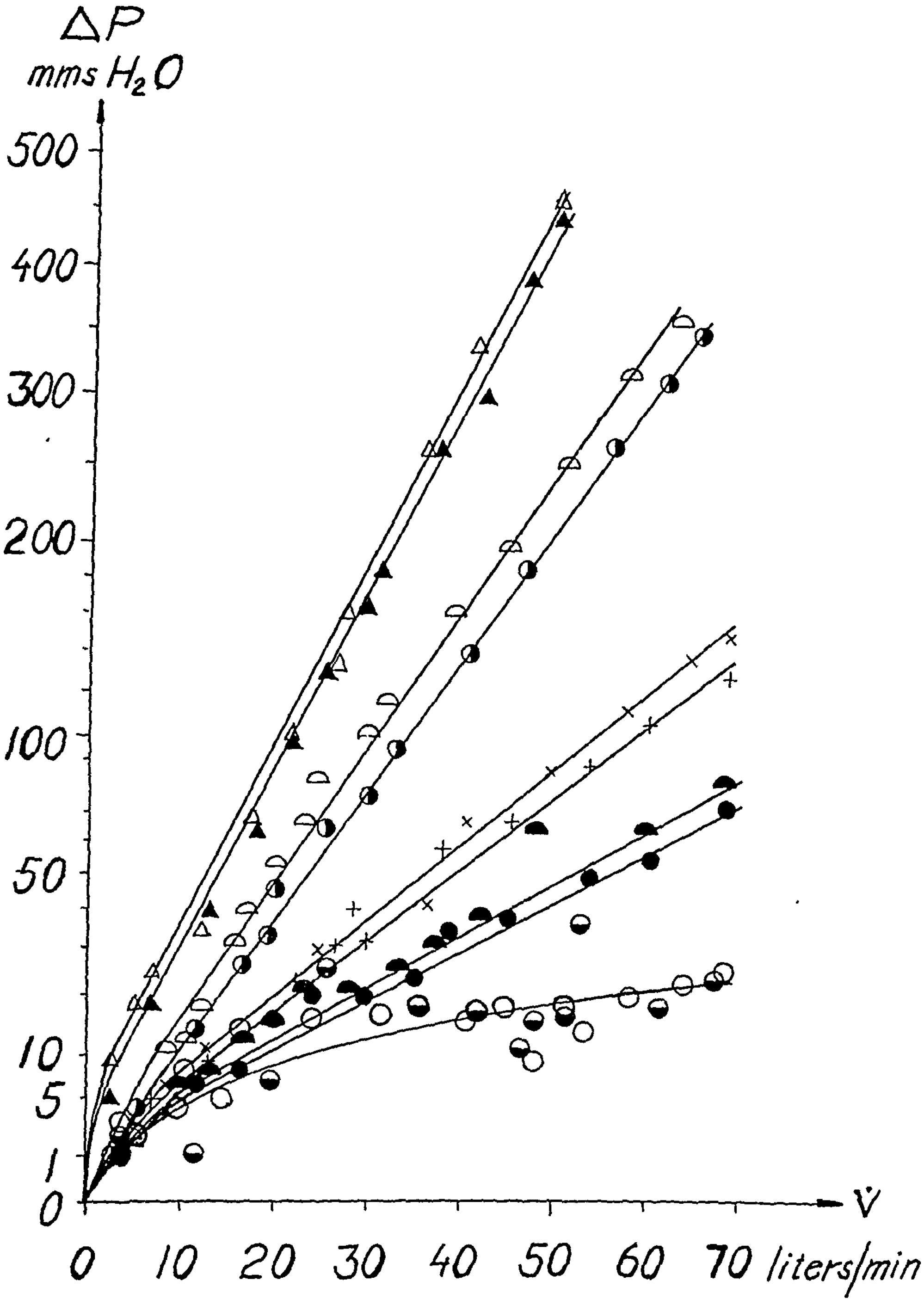


Fig. 3.—Flow rate-resistance relationships in various bronchospirometric catheters. Dry air. Data shown in Fig. 2 are plotted with back pressures on a square scale. (See text.)

tubes The findings of Gaensler[1] were confirmed, in that there exists a rectilinear relationship between the square of the flow rate of dry air through the tube and the resistance against it, indicating the presence of turbulent flow However the large size Carlens catheter allowed streamline flow at all flow rates of dry air

2 Moist air, because of its increased density, heightens the tendency to turbulent flow, and thus the resistance to flow, as compared to dry air used in previous investigations Turbulence is thus encountered under conditions of clinical use in even the largest catheters and at the slowest flow rates

3 The Carlens catheters caused less resistance to air flow than the Marsh or Zavod tubes This superiority, with its evident implications to clinical evaluation of comparative unilateral lung ventilation is readily predictable from the larger cross sectional dimensions of their lumina Since, under conditions of turbulent flow, the resistance developed in any catheter lumen is proportional to the fifth power of its radius, small differences in tube design assume considerable importance

## REFERENCES

1 Gaensler, E A, Maloney, J V, Jr and Bjork, V O Bronchospirometry II Experimental Observations and Theoretical Considerations of Resistance Breathing J Lab & Clin Med 39 935, 1952

2 Carlens, E A New Flexible Double Lumen Catheter for Bronchospirometry, J Thoracic Surg 18 742 1949

3 Norris, C M, Long, J, and Oppenheimer, M J Bronchospirography Apparatus and Technique, J Thoracic Surg 17 357, 1948

4 Zavod, W A Bronchospirography I Description of the Catheter and Technique of Intubation, J Thoracic Surg 10 27, 1940

5 Gebauer, P W A Catheter for Bronchospirometry J Thoracic Surg 8 674 1939

6 Marsh, K A Double Lumen Catheter for Bronchospirometry J Thoracic Surg 25 495, 1953

7 Silverman, L, Lee, G, Plotkin, R, Sawyers, L S, and Yancey, A R Air Flow Measurements on Human Subjects With and Without Respiratory Resistance at Several Work Rates, AMA Arch Indust Hyg 3 461, 1951

Poiseuille's law as long as the pressure exerted on the gas remains the same. The law states:

$$\Delta P = \frac{8\,\dot{V} l\,\mu}{\pi\, r^4}.$$

Where $\dot{V}$ is the flow rate, l is the length of the tube, $\mu$ is the viscosity of the gas, and r is the tube's internal radius.

Of particular importance is the exponential relationship of the resultant back pressure to the radius of the tube; this means that any decrease in tube width (internal diameter) greatly increases the resistance to a given flow. Also noteworthy is the *linear* relationship between pressure and volume flow rate during laminar flow.

However, the departures from the conditions of this law which pertain in the human airway cannulated with a double lumen catheter are significant. (1) *The airways leading from the patient to the spirometers are not straight* but curve gently at the pharynx and in the tubing and adapters leading to the spirometers. At each opening of the catheter and at each adapter between the tube and the spirometer, orifice turbulence can also arise with resultant increase in total resistance. (2) The flow rate of the gas is not uniform but may range during each respiratory cycle from 50 L. per minute in one direction (inspiration) to 40 L. per minute in the opposite direction (expiration).

Except for the slowest flow rates in the largest tubes studied, the relationship between flow and back pressure is not rectilinear (see Fig. 2), indicating the existence of turbulent flow. The Fanning equation for back pressure resulting from turbulent flow states:

$$\Delta P = \frac{f \cdot v^2 \cdot l}{r} = \frac{f \cdot \dot{V}^2 \cdot l}{\pi^2 \cdot r^5}$$

Where v is the linear velocity $= \frac{\dot{V}}{\pi\, r^2}$, f is a friction factor, and $\Delta P$, $\dot{V}$, l, and r are as defined previously.

Thus, the relationship between back pressure and turbulent flow rates is a square one; this effect of turbulence is shown in the curves of Fig. 3 at the higher flow rates (except for the Carlens 41 lumina). The internal dimensions of the tube are seen to assume an even higher exponential (fifth power) relationship to the back pressure developed than in nonturbulent flow.

It should also be stressed that moist air, because of its increased density in comparison with dry air used in previous studies, heightens the tendency toward turbulent flow and thus increases the resistance encountered. As seen in Fig. 4, the flow in the catheters is always turbulent at the flow rates encountered and when moist air is present in the closed respiratory system. The importance of using the largest possible catheter lumina is thus clearly seen.

## CONCLUSIONS

1. Resistance to air flow was measured in the lumina of the Zavod, Marsh, and Carlens (3 sizes) bronchospirometric catheters. The range of flows in the experiments was that which had previously been found in clinical use of the

There has been some discussion about what to call this tracheal bronchus Boyden[3] employs the term "pre-eparterial" for bronchi that arise from the trachea or from the right primary bronchus proximal to the right upper lobe bronchus Foster Carter[4] uses a nomenclature that requires an exact anatomic analysis of the segments of the lung involved When one of the segments or sub segments of the right upper lobe, usually the apical segment, is displaced onto the trachea, he refers to it as a "displaced bronchus", if the right upper lobe has all its normal segments, the tracheal bronchus is called a "supernumerary bronchus"

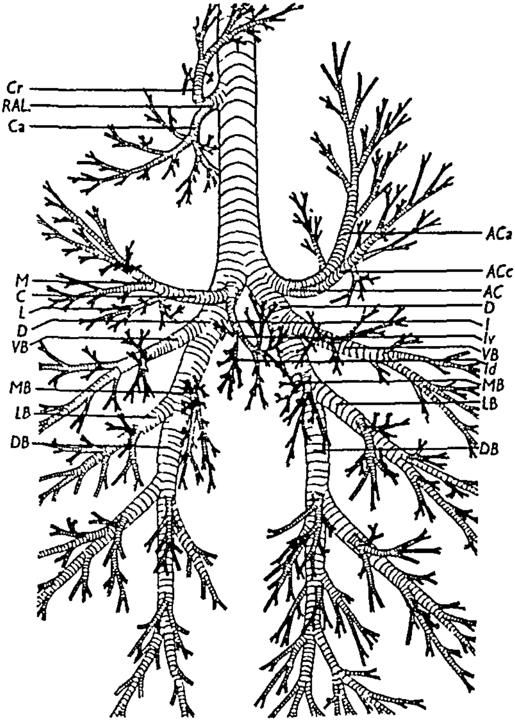


Fig 2—After Hare Ventral view of the bronchial tree in the sheep showing the right apical lobar bronchus (RAL) (Published by permission of the J Anat )

The "displaced bronchus" is a common anomaly, the supernumerary bronchus occurs but rarely Herxheimer[5] in 1901 reported a true supernumerary "third lung" stemming from the right side of the trachea, Mueller[6] described a similar case in 1918 Brock[7] and others have seen additi of a "supernumerary bronchus" On the other hand, the reports

# THE TRACHEAL BRONCHUS

BURNETT SCHAFF, M.D., AND GEORGE BAUM, M.D.
CORAL GABLES, FLA.

IN many mammals a bronchus arises directly from the right side of the trachea; in man this is an anomaly. Flint[1] made an extensive study of the embryology of the pig's lung and beautifully illustrated this tracheal bronchus, which he called Lateral 1 (Fig. 1). Hare[13] demonstrated a similar bronchus in a sheep's lung (Fig. 2). Bremer[2] found that about 5 per cent of human embryos over the 5 mm. size showed one or occasionally two tracheal outpocketings, usually on the right. He reasoned that they probably regressed since these bronchi are rare in the newborn infant.

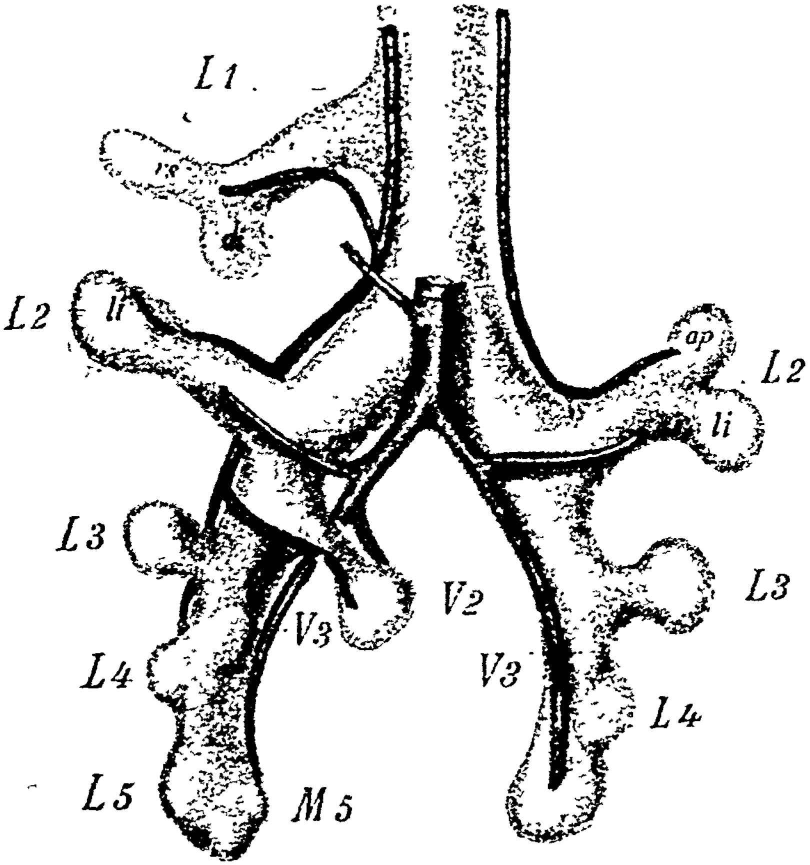


Fig. 1.—Reconstruction of the bronchial tree of a pig 13.5 mm long. (From original of Fig 15, J. M. Flint, Am. J. Anat. 6: 1-138, 1906.)

---

From the Veterans Administration Hospital, Coral Gables, Fla., and the University of Miami School of Medicine.
Received for publication Feb. 15, 1956.

and Neuhauser[11] reported on a patient with a tracheal bronchus with marked irregular stenosis between the tracheal bifurcation and the emergence of the upper lobe bronchus. One of our patients was admitted because of hemoptysis secondary to bronchiectasis in the anomalous bronchus. Some tracheal bronchi are asymptomatic (Gerson and Rothstein,[12] and Brock[7]).

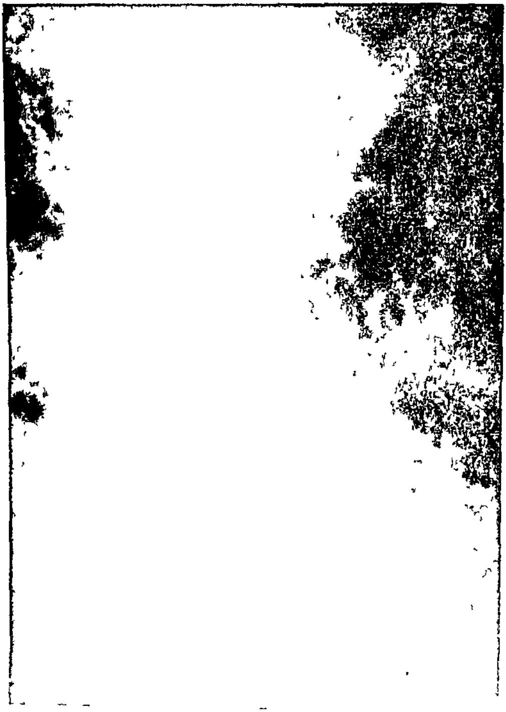


Fig 4.—Photograph of a bronchogram showing a bronchus arising from the trachea

This is a report of two men with a bronchus arising from the trachea; the diagnosis was made by bronchoscopy and confirmed by bronchography In each individual, the right upper lobe showed no apical segment. The proximal branches thus are examples of a "displaced" apical segment. One of the patients presented himself because of hemoptysis. The bleeding came from an aberrant bronchus that was abruptly narrowed at its site of origin from the trachea, (Fig. 3); distally bronchiectatic dilatations were visible. The second patient (Fig. 4) was being investigated because of a so called "Middle lobe syndrome."

have been more numerous. Boyden[3] found one "displaced bronchus" in 400 specimens. Holinger,[8] Huizinga and Smelt,[9] and Brock,[7] mentioned in our bibliography, have observed at least one example of this anomaly.

Holinger and his associates[8] have suggested a simple and useful classification:

1. Normal right upper lobe with 3 normal segments arising above the bifurcation of the trachea.
2. Normal right upper lobe. Abnormal bronchus arising from the trachea.
3. The tracheal evagination consists of only the apical segments (most common).

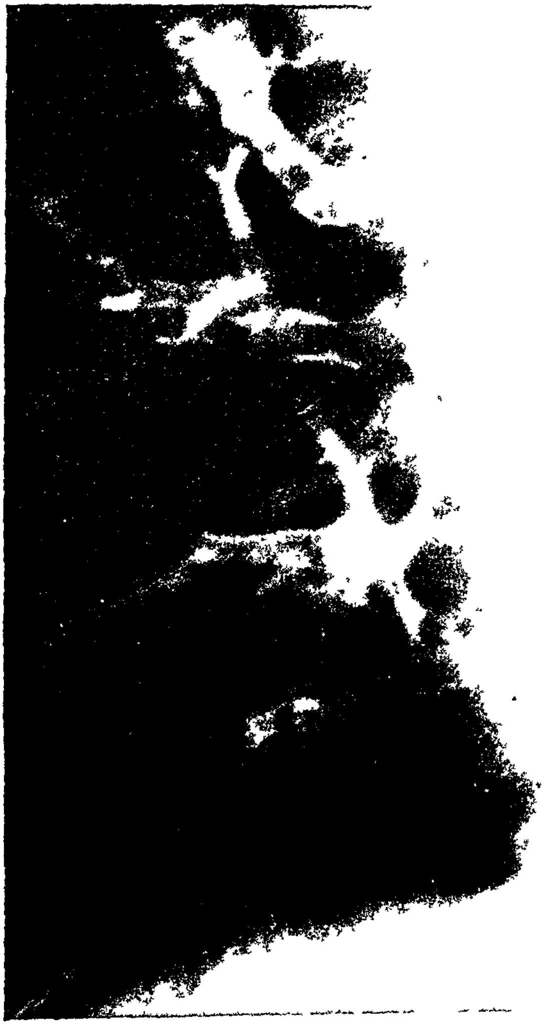


Fig 3.—The bronchus arising from the trachea is narrowed at its site of origin.

The pre-eparterial bronchus appears to be associated with an increased frequency of disease in the area of the lung it aerates. Brock[7] studied one case of supernumerary bronchus with bronchiectasis and one with congenital cystic disease in the right lung. Epstein[10] observed a bronchial adenoma in his patient. Holinger[8] noted that one out of his four patients had symptoms. Ferguson

The Journal of Thoracic Surgery

Vol. 33 March, 1957 No. 3

# Original Communications

## PULSION PHARYNGEAL DIVERTICULA

Ivan N. Ingram, M.D., San Francisco, Calif., and R. P. Foster, M.D.,
Alexandria, La.

ALTHOUGH it has been 191 years since the first case of pharyngeal diverticulum was described by Ludlow in 1764,[3, 23, 24] there is still confusion in the literature concerning the exact site of origin of these diverticula. The present discussion is based upon a review of the literature, a study of hospital records, and personal experience.

The best classification of pharyngeal diverticula is that of Hill[12] (1) Congenital centrally placed anterior diverticula at the base of the tongue (1
(2) Congenital lateral postfaucial diverticula, or aeroceles represent
sistent visceral pouches of the embryo (inner end of the second p
These diverticula usually have a small opening behind the tonsil on the
pharyngeal fold but the opening may become enormous and are often
ciated with a bronchial fistula or cyst.[12]

Pulsion diverticula or deep pharyngeal pressure diverticula, the
common variety is the subject of this report (retrocervical diverticula or pharynx). The opening of these pouches is always posterior in the midline the junction of the upper oblique and lower transverse sphincteric fibers of the inferior constrictor muscle. The latter is known as the cricopharyngeus muscle. There is normally a slight gap between these two sets of muscle fibers known as Killian's dehiscence (Hill[12]). At this site, a depression, the pharyngeal dimple, is described on endoscopic examination. One explanation is that a bolus of food, shot off the back of the tongue in swallowing, impinges upon this part of the pharynx causing the dimple. Marked increase in this recess results in pulsion diverticulum. As the pouch enlarges, it usually extends to the left, but occasionally to the right. A prominent swelling may be noted in

---

Received for publication Jan. 3, 1956.

## SUMMARY

In man, a bronchus arises rarely from the right side of the trachea. Two patients are presented with this anomaly, both being examples of a displaced apical segment.

Philosophers may take delight in noting that man has lost a bronchus that is present in the sheep and the pig, but what is man's place in nature if that also places him in the class with the cat, the rat, and reptilia?

## REFERENCES

1. Flint, J. M.: The Development of the Lungs, Am. J. Anat. 6: 1-138, 1906.
2. Bremer, J. L.: Accessory Bronchi in Embryos, Their Occurrence and Probable Fate, Anat. Rec. 54: 361-374, 1932.
3. Boyden, E. A.: Segmental Anatomy of the Lungs, New York, 1955, McGraw-Hill Book Company, Inc.
4. Foster-Carter, A. F.: Broncho-Pulmonary Abnormalities, Brit. J. Tuberc. 40: 111-124, 1946.
5. Herxheimer, G.: Ueber einen Fall von echter Neben Lunge, Centralbl. f. allg. Path. u. path. Anat. 12: 529-532, 1901.
6. Mueller, H.: Ueber Lappungsanomalien der Lungen, inbesondere über einen Fall von Trachealer Nehen Lunge, Virchows Arch. f. path. Anat. 225: 284-299, 1918.
7. Brock, R. C.: The Anatomy of the Bronchial Tree, New York, 1954, Oxford University Press.
8. Holinger, P. H., Johnston, K. C., Parchet, V. N., and Zimmerman, A. A.: Congenital Malformations of the Trachea, Bronchi and Lungs, Ann. Otol. Rhin. & Laryng. 61: 1159-1180, 1952.
9. Huizinga, E., and Smelt, G. J.: Bronchography, Assen, Netherlands, 1949, Van Gorcum and Co., Ltd.
10. Epstein, I.: Bronchial Adenoma in a Supernumerary Tracheal Lobe, J. Thoracic Surg. 21: 362-369, 1951.
11. Ferguson, C. F., and Neuhauser, E. B.: Congenital Absence of the Lungs (Agenesis) and Other Anomalies of the Tracheo-bronchial Tree, Am. J. Roentgenol. 52: 459-471, 1944.
12. Gerson, C. E., and Rothstein, E.: Anomalous Tracheal Bronchus to the Right Upper Lobe, Am. Rev. Tuberc. 66: 686-690, 1951.
13. Hare, W. C. B.: The Broncho-pulmonary Segments in the Sheep, J. Anat. 89: 387-402, 1955.

specimen is in the Hunterian Museum in Glasgow. Moynihan[24] was impressed with the excellence of the original illustrations and the "accuracy of the description of the symptoms and morbid anatomy."

Monro of Edinburgh, in 1811, described these pouches and discussed the method of their formation (Moynihan, 1927[24]).

Sir Charles Bell,[3] in 1816, presented a patient upon whom he had passed a bougie at intervals of two weeks for a considerable period of time because of the gradual accumulation of mucus in the throat. When this patient died of another cause, the patient's friends asked Bell to examine the body. He found a bag projecting from the lower and posterior portion of the pharynx pushing into a "space betwixt the esophagus and spine." In reviewing Mr. Ludlow's report, Bell[3] stated that Ludlow " . . . believed the bag to be muscular. Although it be hazardous to form a conclusion from a single example, yet in saying the bag is not muscular, I am supported by very strong analogies. I think that the bag is produced by a protrusion or hernia of the inner coat of the pharynx through the fasciculi of the constrictor pharyngis as in diverticula of the urinary bladder." He described the difficulties he had had in passing the bougie and after doing the autopsy could explain the reasons. "It was evident that this bag was the cause of the obstruction by receiving the end of the bougie. For when the instrument was directed backwards, the point of it must have entered the bag, and when carried in the opposite direction, it must have passed into the chink of the glottis. When the bougie was directed laterally, it escaped both these holes and went into the proper canal."

Zenker and Ziemssen[36] first stated that these pulsion diverticula developed at the junction of the pharynx and esophagus. This area is known as the Lannier-Haeckermann area, although Moynihan, in 1927,[24] in searching for Lannier found that the name was Laimer, "a former prosector in Graz." He gave the following reference, Laimer: Med. Jahrbuch Wien, 1888, P. 333.*

Haeckermann's thesis[9] was published in 1891. This area is described as triangular, lying below the inferior constrictor muscle on the posterior wall at the origin of the esophagus, where the musculature was considered weak. Waggett and Davis,[33] however, reported, in 1912, that they examined specimens of gullets from recent autopsies, and performed pressure experiments on them by attaching one end to the tap and securing the other. There was a uniform dilatation of the esophagus above and below the circular and sphincter portion of the inferior constrictor muscle but no weakness could be demonstrated on the wall of the esophagus immediately below the inferior constrictor. Small incisions were made through the longitudinal muscle coat and prolonged water pressure applied, but neither sacculation nor dilatation occurred at the incisions, showing that the remaining portion of the wall of the gullet was strong enough to resist any protrusion of the mucosa. Although subsequently overlooked, Hill,[12] in 1926, reported that he had repeated and confirmed Waggett and Davis' observations.

---

*This reference was obtained at the Stanford Lane Library but the volume did not contain the article. The librarians at Stanford Lane Library thinking that this might be a typographical error found the correct year to be 1883.

the neck when a large distended sac is present and, as will be emphasized later, the diverticulum may extend, in rare cases, into the thorax as far as the lung root.

Moynihan[24] described another form, the lateral diverticulum that he thought differed from the median pulsion diverticulum. Although he had never dissected a specimen of lateral diverticulum, from the drawings and description of the only two records of which he was aware (Butlin[6] and Chevalier Jackson[13]), he was of the opinion that they occur. Mr. Urquhart, at that time (1927) Curator of the Museum at St. Thomas' Hospital in London, agreed with Moynihan that the origin of the diverticulum placed in St. Thomas' Hospital by Butlin was lateral. Moynihan[24] stated that if the pharynx is dissected from behind, a large pad of fat is found at the upper part of the esophagus on each side just below the border of the cricopharyngeus. On teasing away the fat, a gap is found on either side for the passage of the recurrent laryngeal nerve, a branch of the inferior thyroid artery and vein, and a bundle of lymphatic vessels. It was here that he said he would expect a lateral pouch to develop.

Harrington[10] studied, at autopsy, 6 patients with pulsion diverticula who had died of other causes. The defect in 3 was described in the left-posterior wall of the pharynx between the inferior constrictor and cricopharyngeus muscles. In 1, it was in a similar position on the right side, and in 2 cases, the defect was in the cardinal posterior midline through the lower fibers of the cricopharyngeus muscle. If these defects arise laterally, they are certainly rare. Although Harrington titled his article, "Pulsion Diverticulum of the Hypopharynx at the Pharyngo-Esophageal Junction," he described them as originating in the pharynx.

Alexander Lee McGregor, lecturer on surgical anatomy at the University of Witwatersrand, in 1932, published a little book called "A Synopsis of Surgical Anatomy,"[21] in which a chapter on "The Anatomy of Congenital Errors" differentiated congenital from acquired diverticula by their characteristics.

In *congenital diverticulum,* the wall contains all the coats of the parent viscus.

In *acquired diverticulum,* there is little or no muscle in the wall. The neck is narrow and although the diverticulum may be covered by a few muscular fibers, these are insufficient to empty the diverticulum completely. Thus, stagnation of substance passing into it may occur. As the diverticulum enlarges, the neck is smaller than the diverticulum so that the isthmus may become obstructed. His thesis was that these diverticula are dangerous in that they may distend, obstruct, or perforate.

## HISTORY

Mr. Ludlow, surgeon at Bristol,[3, 24] first described pharyngeal diverticula in a letter to Dr. Wm. Hunter as "preternatural dilatation of, and bag formation in the pharynx." The case was observed by Ludlow in 1764, and the between the prevertebral and pretracheal layers of the cervical fascia and may eventually occupy the posterior mediastinum. The sac is usually pyriform in shape as it enlarges and the pharyngeal orifice is fairly wide. As the sac sags down with increasing size, the pharynx comes into alignment with it and the opening of the esophagus appears as a longitudinal narrow aperture on the anterior border of the neck of the sac. This was apparent to Zenker and Ziemssen in 1877.[36] It is thus easy to understand how all food and all instruments pass more readily into the diverticulum than into the esophagus.

There may be variations in the thickness of the sac. It is surrounded by *varying amounts of loose fasciculi of striated muscle arising from the inferior* constrictor. Killian[16] (in 1908) stated that, in the previous ten years, he had observed 7 cases of pulsion diverticula of the pharynx, 6 of which were described by his pupil, von Eichen. Killian described the use of the esophagoscope in making the diagnosis. He noted that the tube usually entered the diverticulum first and then by pulling backward slightly and pressing the tip forward against the cricoid, the opening into the esophagus could be recognized. This is well illustrated in Lahey and Warren's article[18] (illustrations, page 5—the third stage of development). (Fig. 1.) It explains the danger of "blind bougienage" and also how perforation can occur with the esophagoscope. Killian described introducing a small "sonde" and probing behind the larynx until the opening is found and introducing the esophagoscope over the "sonde" and then pulling the latter out. He stated, "a new instrument made by my assistant, Bruinings, offers the combination of the 'sonde' and the tube called 'open beak.' "

Professor Arthur Keith[15] in a paper entitled "A Demonstration on Diverticula of the Alimentary Tract of Congenital or of Obscure Origin," delivered at a meeting of the Royal College of Surgeons of England and published in the *British Medical Journal,* Feb. 12, 1910, presented 10 cases of retrocervical diverticula of the pharynx. Because of the importance of this paper to this subject, it is freely quoted:

> In 6 of the ten cases, the sac is shown in relation to the pharynx. The pouch springs from the posterior wall of the pharynx, its origin situated opposite the cricoid cartilage. The sac is composed of mucous and sub-mucous coats of the pharynx which have gradually evaginated between two definite parts of the inferior constrictor muscle. On smaller sacs and round necks of larger ones may be seen scattered muscle fibers derived from the inferior constrictor. The two parts of the inferior constrictor are separated by the opening orifice of the diverticulum (two figures shown). The lowest part is mainly attached to the cricoid cartilage and serves as a sphincter to the upper orifice of the esophagus. The upper or chief part of the inferior constrictor serves another purpose. Its main function is to seize a bolus of food and force it into the esophagus in the following manner. In deglutition, the larynx is drawn upward and forward, the quadrate plate of the cricoid cartilage being thus drawn away from the posterior wall of the pharynx, so that the bolus of food may sink into the space between the quadrate plate of the cricoid and the inferior constrictor muscle. The moment the bolus reaches the retro-circoid space, it is seized by the inferior constrictor, compressed against the cricoid quadrate plate and forced past the sphincter into the esophagus. Apparently in the act of swallowing the upper sphincter of the esophagus is relaxed, but of the nervous mechanism we know

Killian[16] established this pouch as a protrusion of the mucous membrane of the pharynx, 1 cm. above the origin of the esophagus, between the oblique and transverse fibers of the inferior constrictor muscle in the midline, posteriorly.

As noted above, with enlargement of the diverticulum, it falls to one side of the midline, more often to the left but occasionally to the right. It lies

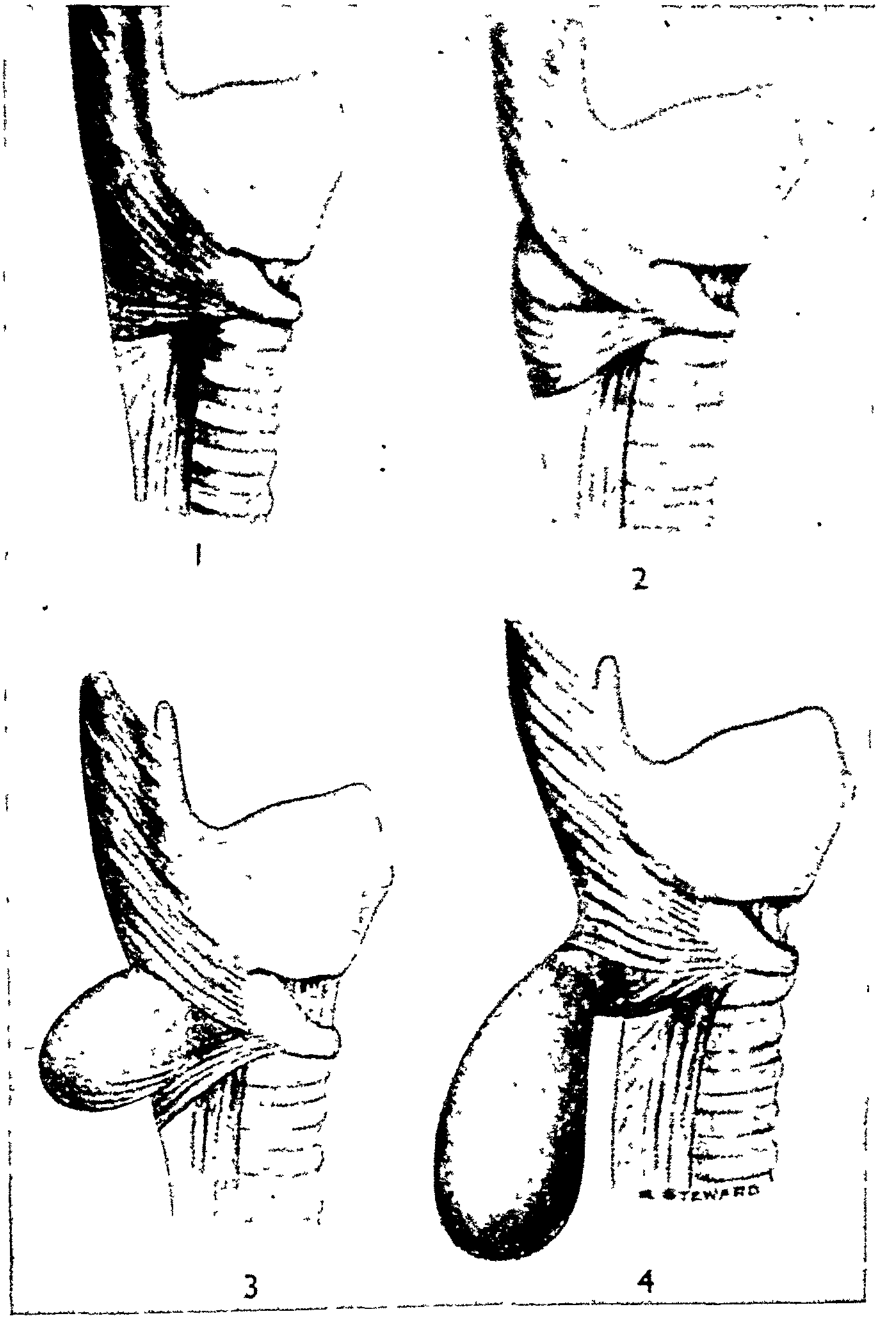


Fig. 1.—Pharyngeal diverticula. *1*, Normal musculature of hypopharynx, showing the arrangement of inferior constrictor muscle *2*, Separation of circular and oblique divisions of the inferior constrictor *3*, Small pouch. *4*, Large pouch (From Negus, V E · Pharyngeal Diverticula, Brit. J. Surg., October, 1950, Fig. 196, page 140 )

In lower animals, the cricoid cartilage lies opposite the base of the skull, but in man it is opposite the sixth cervical vertebra. The sense of smell is most important to the carnivore and herbivore.[25] Mouth breathing is prevented so that all inspired air passes through the nose. The larynx approaches the nasopharynx and the interval anteriorly is closed by a large epiglottis, Negus (1925).[25] Man must rely upon sight because his sense of smell is weak.

In the dog tribe, the epiglottis with the short broad posterior palatine folds acts as a valve so that all air must be inspired through the nose with its extensive olfactory mucous membrane. Some of this air, however, is expired through the mouth when the animal pants, allowing evaporation from the tongue.

There is a space between the epiglottis and soft palate. Changes in the arrangement of the constrictors of the pharynx depend upon the position of the larynx. The inferior constrictor muscle arises from the thyroid and cricoid cartilage and in man they course obliquely to reach the median raphe. The median raphe is attached above to the occiput so downward displacement of the constrictors does not occur. In 1925, Negus[25] gave the most logical reason for the presence of the powerful cricopharyngeal sphincter:

> During respiration, the laryngeal aperture and glottis are open while the mouth of the oesophagus is closed. If this were not so, as the oesophageal opening when relaxed is wider than the glottis, more air would enter the gullet than the lungs The sphincter when contracted exerts pull on its site of origin from the cricoid cartilage and thus holds back the larynx with considerable force during the resting phase At the beginning of the second stage of swallowing, a sensory stimulus originating in the upper postpharyngeal wall is transmitted to the crico pharyngeal sphincter and leads to inhibition and relaxation when a peristaltic wave descends from the naso pharynx to the hypopharynx

The pharyngeal force pump propels a bolus of food to the mouth of the esophagus when the sphincter is relaxed allowing the bolus to pass into the esophagus

> When the sphincter relaxes, the larynx is released It first tilts forward and then moves in an oblique direction upward and forward toward the hyoid bone and mandible As the larynx rises, its aperture is closed by the contraction of the sphincteric girdle of muscles, the thyro arytenoid, ary epiglottic and inter arytenoid; the last passes under the recurved cartilages of Santorini, which are tilted forward. The anterior wall of the oesophagus is attached to the tips of these cartilages (the cartilages of Santorini) and as the crico pharyngeal sphincter is relaxed during this action, wide opening of the mouth of the oesophagus in a funnel like manner results.
>
> Therefore, the closure of the larynx and the opening of the oesophagus is carried out by a simultaneous muscular process In man, the oesophagus is also partially attached to the back of the cricoid cartilage. The opening of the oesophagus is more restricted than in most mammals in which the corniculate cartilages are large and well formed affording sole support of the anterior wall of the oesophagus and allowing wide range of movement
>
> In dogs, the mechanism is very efficient as fibers of the thyro arytenoid muscle pass backward and downward to blend with the longitudinal fibers of the oesophagus Fig. 193, P.138 (Negus' article[25]). Pull on the oesophagus is directed mainly to the anterior wall

> nothing. A study of the mechanism of swallowing shows why diverticula arise at the junction of the two parts of the inferior constrictor, between the upper part which acts as a force pump and the lower part which is sphincteric in action. It is between these two parts that the mucous membrane of the pharynx becomes evaginated to form diverticula.

Professor Keith[15] stated he was certain that these pouches were unconnected with embryological bronchial clefts because the youngest subject on record at that time was 45 years of age.

> Preparations made by the prosector William Pearson show that the diverticula arise where a special deep layer of the inferior constrictor ends. These deep longitudinal fibers have been described by the late Professor Birmingham. After these demonstrations were delivered, Dr. Scanes Spicer was good enough to draw my attention to a very remarkable paper by Prof. Gustav Killian of Freiberg in *Annales des Malodies de Oreille et de Larynx,* 1908—34, P. 8. In this article Professor Killian gives a full description of the upper sphincter of the oesophagus, of its anatomy, physiology, pathology and relationship to pharyngeal diverticula anticipating me in every point and proving his thesis in a more complete manner than I have done. (Keith.)

In Harrington's[10] series there were 107 men and 33 women, or a little over 3 males to 1 female—which is approximately the ratio in the 28 cases when sex was mentioned in the article by Zesas[37] in 1906.

Negus[25] wrote a very interesting and instructive article on pharyngeal diverticula. The anatomy and physiology involved in swallowing, from the evolutionary viewpoint, is most complete. In animals, without a pulmonary apparatus, swallowing is very simple. A tube surrounded by longitudinal and circular muscles executes peristaltic waves which propel food into the stomach. According to him there is no division into stages as in the lung-breathing species.

At the beginning of the second stage of swallowing, the larynx rises with the pharynx. The thyrohyoid assists in the upward movement of the larynx toward the hyoid bone. The whole pharynx is pulled up over the bolus which cannot retreat because of the action of the tongue and fauces with the nasopharyngeal sphincter. In man, the larynx is large in relation to the pharynx. After the initial raising of the pharynx and larynx, a peristaltic wave is executed by the pharyngeal constrictors and travels downward. It commences in the nasopharynx and ends at the mouth of the esophagus. The bolus is carried through the pharynx by force pump action rather than by the pull of reduced pressure as was suggested by Barclay (1930).[1, 2, 26]

In most animals, fibers of the constrictor muscles are arranged in a circular manner. In man, the pharynx is elongated and the larynx descends into the neck so that the fibers of the constrictors run in an oblique direction. The tongue remains large and instead of lying flat in the mouth, as in the anthrapoid apes, it has a recurved shape so that part of it lies in the pharynx. The larynx is closely applied to the hyoid bone, moves with the tongue and lies low in the neck.

> palate and epiglottis, but (man) has rendered himself liable to disabilities such as acquisition of pharyngeal diverticulum. With the obliquity of the inferior constrictor and the horizontal direction of the crico-pharyngeus there is an unsupported triangular area in the posterior wall. During swallowing, the gap becomes greater with the upward movement of the pharynx and larynx. The palato- and stylo-pharyngeus muscles terminate in the lower pharynx and no vertical fibers reach the oesophagus. The latter, being anchored to the diaphragm, exerts a downward pull, which has greater effect on the posterior than on the anterior wall, since the latter is suspended from the larynx.

Negus recapitulates the factors in the development of a pulsion pharyngeal diverticulum as follows: (a) the low position of the larynx and with it the descent of the mouth of the esophagus; (b) the obliquity of the inferior constrictor muscle in relation to the circular arrangement of the cricopharyngeus; (c) the attachment of the anterior wall only of the esophagus to the larynx and its wide range of motion during swallowing; (d) the active contraction of the cricopharyngeal sphincter and the difficulties of coordination of its relaxation with contraction of the pharyngeal force pump; (e) the lack of longitudinal suspension of the posterior wall of the mouth of the esophagus, and (f) the absence of support at the triangular area in the posterior wall.

As everyone does not acquire a pouch, he excluded the anatomical factors common to all, leaving (d) the physiologic factor that may vary, the coordination of relaxation of the sphincter. Although Keith,[15] in 1910, stated that nothing was known of the nervous mechanism of swallowing, he described very accurately the anatomic and physiologic basis of swallowing. He also accurately located the origin of the pulsion pharyngeal diverticulum in the pharynx between the two parts of the inferior constrictor muscle, the upper force pump and the lower sphincteric portion or cricopharyngeus.

Those of us who were fortunate enough to see the motion picture entitled "Normal Mechanism of Deglutition" produced by the Radiology Section of the University of Rochester and presented at the meeting of The American Association for Thoracic Surgery in Montreal, Quebec, in 1954, can marvel at the accurate observations of Killian[16] who, with his associate, von Eichen, studied the swallowing mechanism radioscopically with bismuth, but found that deglutition was so rapid that observation was incomplete. They observed a series of cases with the mirror (hypopharyngoscopy) when the patients were endeavoring to vomit, and watched the opening and closing of the esophagus.

It is Negus' opinion that a pouch in its early stages is the result and not the cause of dysphagia. He lists the causes as:

1. Chronic inflammation of the cricopharyngeal fold or hypopharyngitis which leads to lack of relaxation of the sphincter. Relief may be obtained by dilatation. A small number of patients may, in addition, have superficial glossitis and the few who also have microcytic anemia are classified under the Plummer-Vinson syndrome.

2. Healed inflammatory lesions of the cricopharyngeal fold leading to contraction and stenosis of the mouth of the esophagus with dysphagia and sometimes the formation of a pouch. Negus thought that dilatation should relieve the symptoms.

Man has the stylo-pharyngeal and palato-pharyngeal muscle which terminate before reaching the mouth of the oesophagus with lack of elevation of the posterior oesophageal wall. The oesophagus is fixed below the diaphragm exerting a downward pull and tends to increase the gap between the circular and oblique fibers of the inferior constrictor muscle during swallowing. [Fig. 2.]

The herbivora have an efficient lateral food channel because of the membranous arcus palato-pharyngeus and can breathe simultaneously with deglutition. The arcus palato-pharyngeus is a fold of mucosa enclosing fibers of the muscles surrounding the aperture of the larynx and continuous in the posterior direction. The larynx is thus encircled and projects into the naso-pharynx while respiration is going on. In man, the palato-pharyngeal fibers enclosed in the posterior pillars of the fauces do not descend as far as the hypo-pharynx and do not form an encircling ledge or fold, nor do they reach the mouth of the oesophagus as in the herbivora to assist in opening the gullet during swallowing.

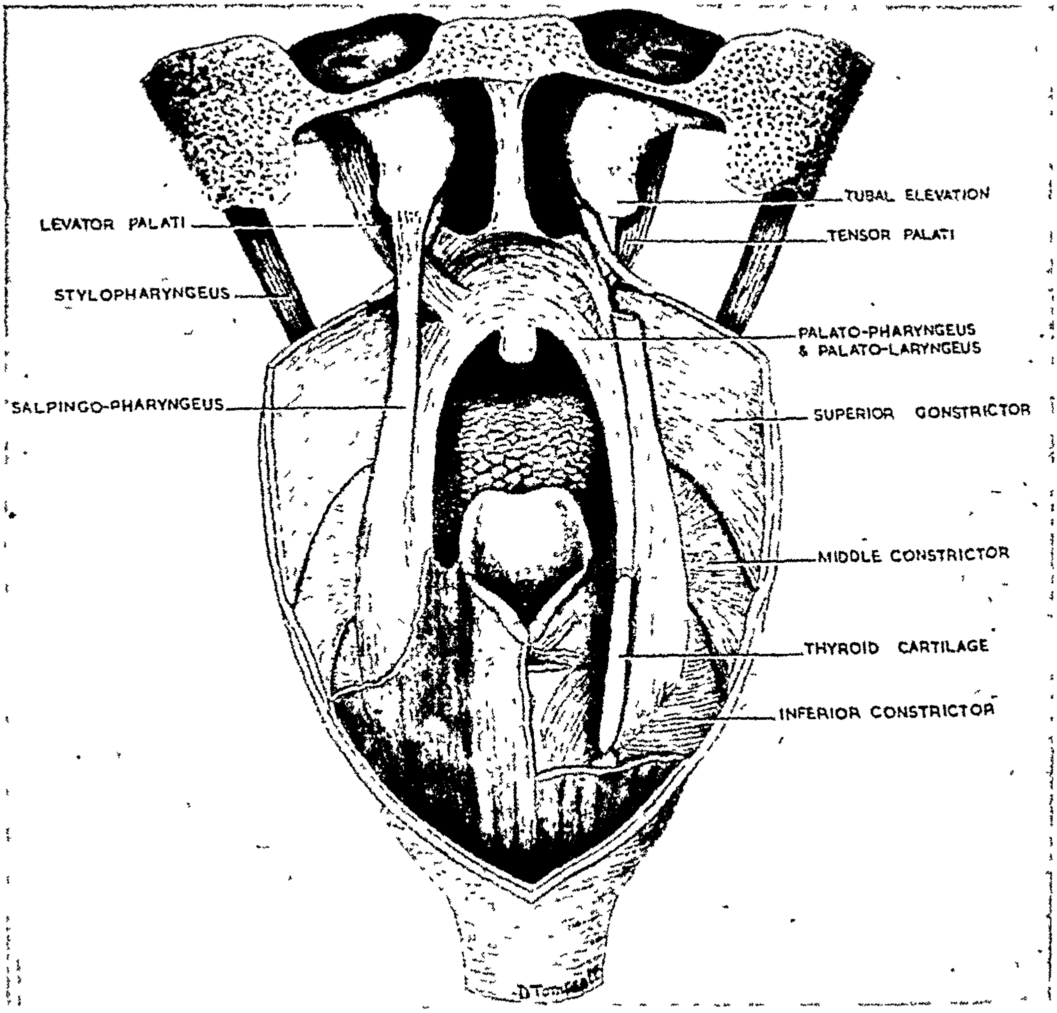


Fig. 2.—Pharyngeal diverticula. Human pharynx open from behind to show the elevator and constrictor muscles (From Negus, V. E : Pharyngeal diverticula, Brit. J. Surg., October, 1950, Fig. 179, page 129.)

Humans gained much from the assumption of an erect posture, from a large pharyngeal resonator for phonation and from separation of the soft closure of the rent in the esophagus with sutures even if this results in only temporary closure of the opening. Antibiotic therapy is, of course, an important adjustment.

Overstreet and Ochsner[28] reported 13 cases of traumatic rupture of the esophagus, 8 of which occurred at endoscopic examination. Five of these perforations were located by them "immediately below the cricopharyngeus sphincter on the posterior wall."

## SYMPTOMS

The symptoms are fairly distinctive: dysphagia is described as intermittent and progressive; regurgitation of undigested food is frequent and may occur during or soon after eating, to several hours or days later; backward motion of the head or pressure upon the neck may propel food back into the mouth after it has been swallowed and lodged in the diverticulum; a swelling may be noted, usually on the left side, enlarging as the meal is eaten so that it may resemble a thyroid tumor.

Moynihan[24] described gas developing in the pouch and escaping into the pharynx producing a condition first described and considered by him pathognomonic. "As the patient talks, movements in the neck cause an expulsion of gas from the pouch, and the words spoken have 'bubbles' in the middle of them or among them; there is a curious but unmistakable spluttering of air in and among the spoken words. The phenomenon may be produced in a very striking manner if the patient is asked to read continuously while a gentle pressure is made upon the neck."

Inhalation of particles of food that are suddenly expelled from the pouch into the pharynx brings about coughing which may be so severe that dyspnea and cyanosis result. When this awakens a patient from sleep, it is naturally very terrifying. Inhalation pneumonia and lung abscess have been described which may be fatal. With increase in the size of the diverticulum, there may be displacement of the esophagus with so much pressure that there is inadequate intake of food to maintain nutrition with consequent weight loss and inanition. Deaths have been reported from starvation.

## DIAGNOSIS

Many writers have emphasized the dangers of perforation when employing blind bougienage and esophagoscopy. Following a good history and physical examination, adequate barium study of the pharynx and esophagus is all that is necessary to make the diagnosis. Lahey[17] cautioned that a semilateral view should be taken, as well as the anteroposterior projection, to rule out a dilatation of the esophagus above an esophageal web. We also take lateral films of the diverticulum and x-rays of the chest are routine. If the diverticulum is huge and there is danger of aspiration of the contents of the diverticulum if it is filled with barium, the use of Lipiodol rather than barium should be considered. The vocal cords are examined preoperatively with the

3. Excessive contraction of the sphincter without evidence of pathologic change. Stretching with the hydrostatic bag may give relief and delay the formation of a pouch.

4. Lack of relaxation at the right moment in the second stage of swallowing may be due to deficiency in the sensory stimulus which originates in the posterior pharyngeal wall from the impact of food. The reflex arc comes through the pharyngeal plexus to the vagal fibers which relax the sphincter. The motor cells from which these fibers originate are situated in the nucleus ambiguus of the spinal accessory nerve. They leave the skull in that nerve and pass, after leaving the jugular foramen, to the ganglion nodosum of the vagus via the internal branch of the accessory (Negus).[26]

Rose[30] reported a large diverticulum in a 69-year-old man with a ten years' history of dysphagia causing complete obstruction to the esophagus. This was presented as "one of the less common causes of dysphagia." This was one of only 2 cases recorded at the Royal North Shore Hospital of Sydney in 25 years. At esophagoscopy, the esophageal lumen was represented by a tiny dimple high upon the anterior wall in the midline. A Wangensteen tube was placed into the opening under direct vision, and passed into the stomach. This was used for feeding. Under general anesthesia, the pouch was removed in one stage, transfixing the neck of the sac with chromicized catgut. The sac measured 7.5 by 7.5 by 5.0 cm. Barium bolus after operation and at intervals of one year and two years, postoperatively, showed a small residual pouch, but there was no increase in the size after two years. He emphasized that a Wangensteen tube should always be inserted under direct vision. In a case reported by Harrington[10] (1945), the sac, extending down into the mediastinum, held 825 c.c. of fluid after its removal. McGregor,[21] and Lahey[17] have stressed that unskilled endoscopy may cause perforation of the pouch.

Bisgard and Kerr[5] presented a very instructive article on the surgical management of instrumental perforation of the esophagus. These perforations occur most frequently during esophagoscopy. The esophagus may be torn in passing the esophagoscope above, at the cricopharyngeal pinch cock of Jackson (cricopharyngeus) or at the cardia, the diaphragmatic pinch cock (right crus of the diaphragm). It may occur in the process of removing a foreign body or in obtaining a biopsy specimen. It is of interest that the flexible gastroscope is the second most common offender. Blind bougienage in the dilatation of strictures of the esophagus is the third most common cause. They present 1 case that was self-inflicted, resulting from passing a stomach tube. In 5 cases cited by them, 3 involved the cervical esophagus. All of these perforations occurred during esophagoscopy. In one of these, followed to autopsy, the diverticulum extended from the pharyngoesophageal "junction" to the lung root with a chicken bone at the neck of the diverticulum that had perforated the left wall of the esophagus. They emphasize that in instrumental perforations, the endoscopist is usually aware that perforation has occurred, and that no time should be lost in establishing drainage, whether the perforation occurs in the cervical area or in the thorax. They also advise no deaths. Temporary pharyngeal fistula occurred in 5 cases and, in 3, hoarseness subsided in one week or ten days. In 1 patient, in whom there was hoarseness before operation, paralysis of a vocal cord resulted with continuing hoarseness. Postoperative dilatation was carried out in 5 patients because of angulation of the esophagus. Average hospitalization was less than two weeks, and the average time until dismissal after operation was three weeks. There was recurrence in only 2 cases. In 1, symptoms improved after dilatation, but it was thought that operation might later be necessary. A second operation was done in the other case. After ten days, a pharyngeal fistula formed which drained for three weeks, but the wound was completely healed in four weeks. There was no mediastinitis. He pointed out that there was little danger of mediasinitis after the one-stage operation because fistulas never developed earlier than seventy-two hours after operation, when the mediastinal fascial planes should be walled off. In 15 of the 115 patients, the diverticula filled the entire mediastinum, but there was no indication of mediastinitis postoperatively in a single case.

Many surgeons prefer local anesthesia (Harrington, 1945[10]) but we feel that the advantages claimed for it are far outweighed by the relaxation and safety afforded by intratracheal ether anesthesia.

The pouch should be approached from the side it presents. In Harrington's 140 patients, the approach was left cervical in 116, and right cervical in 24.

Mortensen, Clagett, Schmidt, and Gray[23] reported their experience with 339 cases treated during the 10-year period, Jan. 1, 1944, through Dec. 31, 1953, by the one-stage method with a hospital mortality rate of only 0.9 per cent.

By the time the sac is large enough to distend and then spill over into the esophagus (the fourth stage of Lahey), posteroanterior and lateral films usually show readily on which side the pouch lies. Occasionally, however, even in fairly large sacs, it is necessary to pass a catheter into the pouch to show its relation to the barium-filled gullet. (Fig. 3.)

The approach is through a longitudinal incision along the anterior border of the sternocleidomastoid muscle beginning above the level of the cricoid cartilage to the medial end of the clavicle. The omohyoid and sternothyroid muscles are retracted medially and the carotid sheath and sternocleidomastoid are retracted laterally. The thyroid gland is retracted medially exposing the fascia investing the trachea and esophagus. The neck of the diverticulum is found at the level of the cricoid cartilage. Esophagoscope and bougies are not necessary to locate a pharyngeal diverticulum and, of course, in the hands of others than the expert, are dangerous. The neck of the sac is separated from the surrounding tissue, transfixed with chromic catgut, and excised. The stump is invaginated into the wall of the pharynx. A Penrose drain is placed into the former bed of the diverticulum, and the incision closed with interrupted catgut sutures, with Dermalon or cotton in the skin. Many surgeons prefer nonabsorbable sutures in closure of the incision, but we feel that, in a potentially infected field, catgut is the suture material of choice. The drain should be removed in three or four days.

mirror and again postoperatively as in thyroid surgery. Moynihan[24] and Lahey[18] both emphasized that a malignant lesion must be suspected when the barium-filled outline of the sac is irregular.

We also like to inspect this region directly, because at times it is difficult and may be impossible to locate the esophageal orifice when it has been displaced anteriorly by the pharyngeal diverticulum. In order to get a catheter into the stomach when a large pouch deflects the esophageal opening anteriorly, the Levine tube must be inserted under direct vision. The patient is fed through the Levine tube for at least one week postoperatively, if he tolerates it.

### SURGICAL TREATMENT

A most complete review of the surgical treatment of pulsion pharyngeal diverticula was given in Lahey's last paper.[18] Niehans[18] has been given credit for the first excision in 1884. In reviewing this case[37] of a man, 57 years of age, with both goiter and diverticulum, it was found that on Aug. 8, 1884, the "left-sided goiter" was removed and on Aug. 26, 1884, the diverticulum was excised with the thermocautery between two ligatures. After eight days, a fistula developed so that milk the patient drank came out of the wound. On the twentieth of September, approximately three weeks from the date of excision of the diverticulum, sudden erosion of the superior thyroid artery occurred with death from hemorrhage. Wheeler[34] reported a case, in 1886, that he had operated upon successfully, on June 13, 1885, in which he removed both a large congenital lateral pharyngeal diverticulum or pharyngocele and a pharyngeal pulsion diverticulum, so he must be given credit for the first successful excision of two of three types of pharyngeal diverticula.

Bergmann[4] operated upon his first patient successfully, in 1890, and re-orted it in 1892. Butlin's[6] first case, also successful, was seen in 1890, and ough operation was advised several months before Bergmann operated upon is patient, Butlin's patient did not submit to operation until June, 1892. In 398, he reported his second operative case, reviewing the 6 cases he had seen t that time. One of these patients, a male aged 57, of Cleveland, Ohio, had reviously been seen by Dr. Richardson of Boston, who advised removal of the ouch. He was seen in consultation by Butlin with Edward F. Cushing of Cleveland. He was included in Richardson's[29] review of his experience in 1900.

Zesas,[37] in 1906, collected a total of 42 cases in which a pulsion diverticulum was excised. Twenty-eight patients were men and 9 were women. The sex was not stated in fiive reports. Primarly healing resulted in 6; fistula developed in 28 but subsequently healed. There were 8 operative deaths.

Lahey[18] presented a total of 365 patients operated upon for pulsion pharyngeal diverticulum with follow-up on 250 for at least two and one-half years. As everyone knows, he was the champion of the two-stage operation. He recorded two deaths in 365 patients and twelve recurrences in 250 cases, or 4.8 per cent.

Harrington,[10] in 1945, presented a comparable number of 140 cases, in which a one-stage procedure was done in 115, and a two-stage operation in 25. In the 115 patients, on whom a one-stage operation was performed, there were

## REPORT OF A TYPICAL CASE

A 45-year-old man of Mexican extraction was admitted to the hospital on March 15, 1954, complaining of choking sensation in the throat toward the end of a meal, with regurgitation of small amounts of undigested food immediately after eating up to several hours later. The onset of symptoms was two years previously and in the six months before admission they had become worse. The patient's wife noted that far into the night the patient would regurgitate undigested particles of food eaten that day. By pressing on either side of the midline above the sternal notch he could elicit a gurgling sound. There had been no nausea but he had lost 9 pounds in two months.

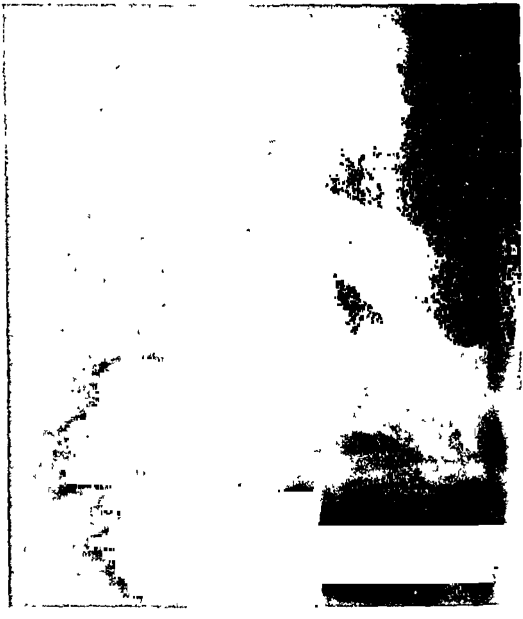


Fig. 4.—After filling of diverticulum, the barium spills into the esophagus.

Past history was negative except for a cholecystectomy performed ten years previously.

He had been married twenty-five years and was the father of four children.

General physical examination was entirely negative. On fluoroscopy, the esophagus would not fill until the diverticulum was distended and spilled over into the esophagus (Lahey's fourth stage). Because the position of the diverticulum in relation to the esophagus could not definitely be determined, a catheter was placed into the diverticulum from above which proved that it sagged to the left side and extended into the mediastinum to the level of the fourth thoracic vertebra (Fig. 3). Fig. 4 shows that after filling of the diverticulum, the barium spilled into the esophagus which was displaced anteriorly and to the right. The vocal cords moved normally. At esophagoscopy

Sweet[32] advises interrupted sutures of fine silk in the mucosa, tied on the lumen side. He makes the point, which is good, that the drain should not impinge against the pharynx at the site of the base of the excised diverticulum.

The Levine tube, preferably placed in the stomach through the nose prior to surgery or under direct vision just before the anesthetic is begun, should be used to feed the patient for the first seven days postoperatively. With this regimen, as in one of our patients, actual weight gain may result. Gastrostomy is unnecessary.

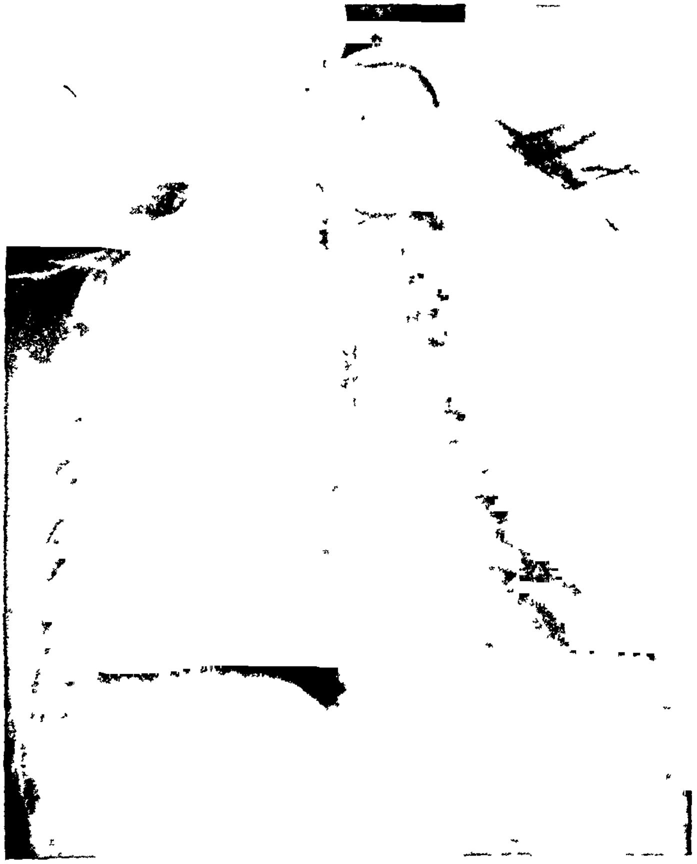


Fig 3.—Shows catheter passed into the diverticulum from above, proving that the diverticulum sags to the left

Johansson and Michas[14] reporting their results in 10 cases of "pharyngo-esophageal diverticula," had no surgical fatality but there was one persistent recurrent laryngeal nerve paralysis and a fistula occurring on the fourteenth day in another. There was evidence of recurrences of the diverticula by x-ray in 4 cases. However, these were merely bulges in the esophageal wall. There were *no* symptoms of recurrences in these patients. They stated that the smallest spur remaining in the repair acts as a nidus for recurrence. They were of the opinion that, by giving water by mouth in the early postoperative period, the suture line would be cleansed and be more favorable than tube feeding.

esophagus, the danger of esophagoscopy and blind bougienage is readily apparent In 1 of our patients, it was impossible to see the opening of the esophagus under direct vision with the laryngoscope so that a Levine tube could not be passed beyond the diverticulum into the esophagus, preoperatively

The important factor in the development of a pulsion pharyngeal diverticulum, as emphasized by Negus, is the active contraction of the cricopharyngeal sphincter and incoordination of its relaxation with the active contraction of the upper pharyngeal force pump Symptoms are reviewed The diagnosis depends essentially upon a good history and physical examination and adequate barium or Lipiodol study Preoperative and postoperative vocal cord examination is routine The history of the surgical treatment is reviewed and the advantages of the one stage operation emphasized

Eight operative cases are presented of which the experience in one case is reviewed in detail

## CONCLUSIONS

Pulsion diverticula or deep pharyngal pressure diverticula are entirely pharyngeal in origin, arising from the posterior wall at the junction of the upper oblique and lower transverse sphincteric fibers of the inferior constrictor muscle The physiologic factor of importance is the incoordination of relaxation of the lower transverse sphincteric fibers of the inferior constrictor muscle or cricopharyngeus in relation to the active contraction of the force pump upper oblique fibers of the inferior constrictor muscle

## REFERENCES

1 Barclay, A E Normal Mechanism of Swallowing, Brit J Radiol 3 534 546 1930
2 Barclay, A E Normal Mechanism of Swallowing, Proc Staff Meet Mayo Clin 5 251 257, 1930
3 Bell, Sir Chas Clinical Observations, London, 1816, Longman (and others), pp 64 72
4 Bergmann, E Ueber Oesophagus divertikel und seine Behandlung, Arch klin Chir 43 10 30, 1892
5 Bisgard, J D, and Kerr, H H Surgical Management of Instrumental Perforation of the Esophagus Arch Surg 58 739 751, 1949
6 Butlin, H T On a Second Case of Removal of a Pressure Pouch of the Oesophagus, Brit M J 1 8 10, 1898
7 Buxton, R Possible Etiological Factor in a Pharyngo Esophageal Diverticulum, A M A Arch Surg 62 601 603, 1951
8 Conole, F D, and D'Angelo, A A Resection of Pharyngeal Diverticulum With Spontaneous Development of Tension Pneumoperitoneum, Am J Surg 83 580 583, 1952
9 Haeckermann, K Beit Z Lehre von der Entstehung der Divertikel des Oesoph, Gottingen, 1891, W F Kaestner
10 Harrington, S W Pulsion Diverticulum of the Hypopharynx at the Pharyngo Esophageal Junction, Surgery 18 66 81, 1945
11 Hearn, W J The Removal of an Oesophageal Diverticulum, Tr Am Surg Assn, Phila 17 262 269, 1899
12 Hill, W Pharyngeal and Oesophageal Diverticula, Brit M J 2 1163 1169, 1926
13 Jackson, C, and Shallow, T A Ann Surg 83 1 19, 1926
14 Johansson, L, and Michas, P Diverticula of the Esophagus, A Study of 17 Cases, J THORACIC SURG 27 361 372, 1954
15 Keith, A A Demonstration on Diverticula of the Alimentary Tract of Congenital or of Obscure Origin, Brit M J 1 376 380, 1910
16 Killian, G La boudre de l'oesophage, Ann d mal d' oreille, du larynx (etc ), Paris 34 1 52, 1908
17 Lahey, F H The Management of Pulsion Esophageal Diverticulum, J A M A 109 1414 1419, 1937

local anesthesia on March 12, 1954, the esophagoscope passed directly into the pharyngeal diverticulum. It was impossible to see the orifice of the esophagus beyond. At operation on March 17, 1954, it was impossible to pass a Levine tube beyond the diverticulum under direct vision. The diverticulum was excised through a longitudinal incision along the anterior border of the left sternomastoid muscle. Intratracheal ether was employed. The carotid artery and jugular veins were gently retracted laterally. A small thyroid vein was divided between ligatures and the sac gently retrieved from the mediastinum. Investing fibers of the inferior constrictor muscle about the neck of the sac were sparse. The neck of the sac was cleared of this, transfixed with chromic catgut, and excised at the pharyngeal wall. The stump was inverted into the wall of the pharynx. A Penrose sheath drain was placed into the mediastinum in the region of the former bed of the diverticulum. The incision was closed in layers with interrupted sutures of catgut and Dermalon in the skin. The drain was completely removed on the fourth day. The incision healed per primum. On the first postoperative day, a small polyethylene catheter, 3 mm. in diameter, was placed through the corner of the patient's mouth into the stomach. With this small caliber tube there was no discomfort. It was retained for seven days through which a nutritious diet was instilled into the stomach by continuous drip. During this week the patient gained several pounds in weight.

The specimen received in Formalin in the pathology department measured 3 by 5 by 2 cm. Histologic diagnosis was ulcerative subacute inflammation in a pharyngeal diverticulum. The patient was discharged from the hospital on the eleventh postoperative day. Postoperative films on June 30, 1954, showed no evidence of recurrence and the patient was enjoying his food.

Thirteen other patients with pharyngeal diverticulum have been followed, 7 of whom have been operated upon. The ages varied from 41 to 64. All of these patients were men. The approach was through the left side of the neck in all but 2. A two-stage operation was done in only 1 case. No recurrences have been reported; however, a complication of persistent left recurrent laryngeal nerve paralysis has occurred once.

## SUMMARY

Pulsion diverticula or deep pharyngeal pressure diverticula arise from the posterior aspect of the pharynx at the junction of the upper oblique and lower transverse sphincteric fibers of the inferior constrictor muscle. These are acquired diverticula and are characterized by having little or no muscle in the wall. The neck is narrow and the muscular fibers covering the pouch are insufficient to empty it. These diverticula may distend, obstruct, or perforate. Killian, in 1908, established this pouch as a protrusion of the mucous membrane of the pharynx, 1 cm. above the origin of the esophagus, between the oblique and transverse fibers of the inferior constrictor muscle in the posterior midline. With enlargement of the diverticulum, it falls to one side of the midline, usually to the left. It lies between the prevertebral and pretracheal layers of the cervical fascia and may eventually enlarge so that it occupies the posterior mediastinum. As the sac enlarges and sags down, the pharynx comes into alignment with it and the esophagus appears as a longitudinal narrow aperture on the anterior border of the neck of the sac. As food and instruments pass more readily into the diverticulum than into the

# THE ANGIOCARDIOGRAPHIC DEMONSTRATION OF SUPERIOR VENA CAVAL CONSTRICTION IN CONSTRICTIVE PERICARDITIS

HAROLD A. LYONS, M.D.,* JESSE MINNIS, M.D.,** AND EDWIN GRIFFIN, M.D.***
NEW YORK, N. Y.

THE effective treatment of constrictive pericarditis is designed to liberate all critical areas so that the decortication must extend beyond the left border, right border, and lower border, with release of both the inferior and the superior vena cava and the base of the heart.[1] Although there has been definite involvement of the venae cavae, this constriction has never been definitely demonstrated by any of the diagnostic methods. Except for edema of the face and head noted clinically in a patient in whom constrictive pericarditis and superior vena caval involvement was found at operation,[2] no other report has indicated that this involvement has been demonstrated before operation.[3, 4] It is for this reason that this report on the angiocardiographic demonstration of constriction about the superior vena cava in a patient suffering from constrictive pericarditis is presented. The caval constriction was proved by operation. It is equally important to be able to demonstrate this involvement, for Holman and Willett[5] state that constriction of the vena cava may continue to be a source of trouble when the right heart only is decorticated and actual liberation of the venae cavae is disregarded.

## CASE REPORT

F. V., a 43 year old white married woman, was first admitted to Kings County Hospital on June 3, 1955, with the chief complaint of shortness of breath. She had a three year history of swelling of the legs and dyspnea on exertion. During this time she was under treatment by her family physician, being given daily digitalis, weekly diuretics, and a low salt diet. She stated that she had had a "leaking heart" for many years, but a history of tuberculosis or significant acute infections was lacking. No history of rheumatic fever was obtained. On physical examination, she was found to have a blood pressure of 114/80 mm. Hg, auricular fibrillation, distended cervical veins, and distant heart sounds without murmurs or thrills. A diastolic shock was felt over the apex. Massive ascites and moderate pretibial edema were noted. A venous pressure determination was 270 to 310 mm. water in all extremities, the systemic circulation time was 18 seconds (Decholin). On abdominal paracentesis, 16 L. of straw colored fluid were obtained which did not contain bacteria, tubercle bacilli, or malignant cells. After this procedure, no abnormal abdominal or pelvic findings were found. Fluoroscopic and roentgenographic examination of the chest showed

---

From the Department of Medicine, State University of New York College of Medicine at New York City, and the Thoracic Surgical Department, Kings County Hospital Center, Brooklyn, N. Y.

Received for publication Jan. 27, 1956.

*Associate Professor of Medicine, State University of New York College of Medicine at New York City.

**Resident, Thoracic Surgery, Kings County Hospital Center at Brooklyn, N. Y.

***Director, Thoracic Surgery, Kings County Hospital Center at Brooklyn.

18. Lahey, F. H., and Warren, K. W.: Esophageal Diverticula, Surg., Gynec. & Obst. 98: 1-28, 1954.
19. Laimer, E.: Beitrag zur Anatomie des Oesophagus, Med. Jahrb. Wien, 1883, Wilhelm Braumuller, pp. 333-388.
20. Mayo, C. H.: Diagnosis and Surgical Treatment of Oesophageal Diverticula: Report of 8 Cases, Ann. Surg. 51: 812-817, 1910.
21. McGregor, A. L.: A Synopsis of Surgical Anatomy: The Anatomy of Congenital Errors, New York, 1932, Wm. Wood & Company, pp. 303-382.
22. McNealy, R. W., and Glassman, J. A.: A One Stage Pharyngo-esophageal Diverticulectomy, Surgery 21: 470-475, 1947.
23. Mortensen, JD, Clagett, O. T., Schmidt, H. W., and Gray, H. K.: One-Stage Pharyngo-esophageal Diverticulectomy, J. THORACIC SURG. 30: 316-322, 1955.
24. Moynihan, Sir Berkeley: Harveian Lecture on Diverticula of the Alimentary Canal, Lancet 1: 1061-1066, 1927.
25. Negus, V. E.: Evolutionary Factors in the Production of Pharyngeal Diverticula, J. Laryng. & Otol. 40: 702-724, 1925.
26. Negus, V. E.: Pharyngeal Diverticula: Observation on Their Evolution and Treatment, Brit. J. Surg. 38: 129-146, 1950.
27. Netterville, R. E., and Harris, E. J.: Esophageal Diverticula, Mississipi Doctor 29: 296-300, 1952.
28. Overstreet, J. W., and Ochsner, A.: Traumatic Rupture of the Esophagus: With a Report of 13 Cases, J. THORACIC SURG. 30: 164-180, 1955.
29. Richardson, M. H.: Two Cases of Oesophageal Diverticulum With Remarks, Ann. Surg. 31: 525-546, 1900.
30. Rose, T. F.: Pharyngeal Diverticulum: Report of a Large Diverticulum Causing Complete Obstruction to the Oesophagus, M. J. Australia 2: 776-779, 1951.
31. Slobodkin, M.: Goitre Plongeant (Plunging Goiter) Associated With Pharyngo-Esophageal Diverticulum, Radiology 58: 378-382, 1952.
32. Sweet, R. H.: Pulsion Diverticulum of the Pharyngo-Esophageal Junction: Technic of the One Stage Operation. A Preliminary Report, Ann. Surg. 125: 41-48, 1947.
33. Waggett, E. B., and Davis, E. D.: A Case of Pharyngeal Diverticulum, Lancet 1: 786-788, 1912.
34. Wheeler, W. I.: Pharyngocele, Dublin J. M. Sc. 81: 460-461, 1886.
35. Wilkie, D. P. D., and Hartley, J. N. J.: Pharyngeal Diverticulum and Its Surgical Treatment, With a Record of 2 Cases, Brit. J. Surg. 10: 81-92, 1922.
36. Zenker, F. A., and Ziemssen, H.: Krankheiten des Oesophagus, in Handbuch der Speciellen Pathologie und Therapie 7: (Supp.) Leipzig, 1877, Vogel, pp. 50-87.
37. Zesas, D. G.: Beitrag zur Chirurgischen Behandlung des Speisenröhren Divertikels, Deutsche Ztschr. f. Chir. 82: 573-594, 1906.

A constricted area around the superior vena cava at its junction with the right atrium was demonstrated in the angiocardiogram (Figs 3A and 3B) and both right and left auricles were enlarged A normal sequence of filling of all chambers was present (Fig 4) Small areas of calcification were observed in the vicinity of the right atrium

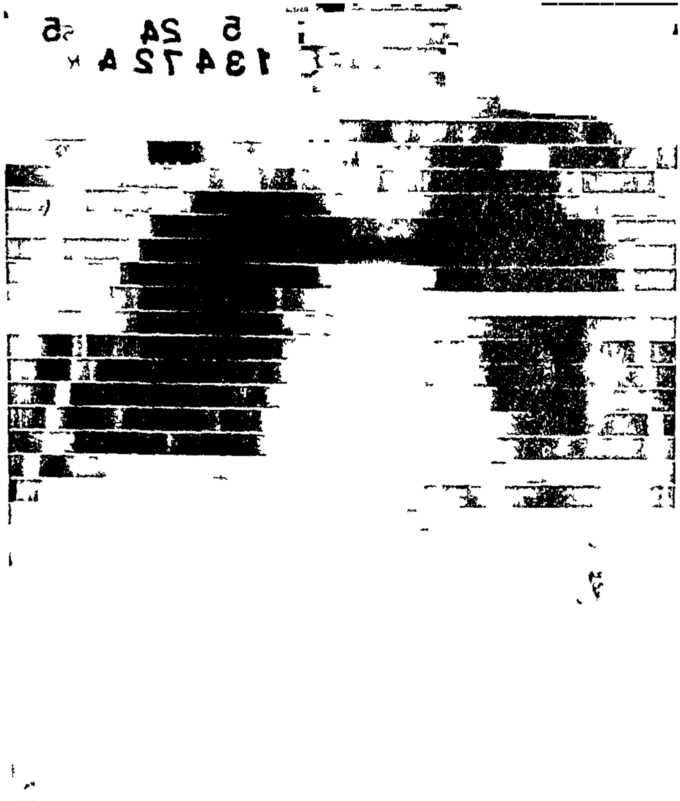


Fig 2—The roentgenkymogram showing the typical findings of constrictive pericarditis of rapid ventricular filling (diastolic dip) and diastolic plateau

On Aug 4, 1955, the patient was operated on for decortication of the pericardium The procedure was performed from "phrenic to phrenic" as described by Delorme[5] and later popularized by Churchill[6] The pericardium contained areas of calcifications one fairly large area of calcification was located, significantly, just anterolateral to the right auricular appendage Dissection and resection of the pericardial "peel" was carried about this calcific plaque leaving it in situ Just cephalad to this area, at the junction of the moderate enlargement of the heart. The right ventricle and left atrium were believed to be enlarged (Fig. 1). The electrocardiogram showed auricular fibrillation, low voltage in all leads, a normal axis, and an occasional premature ventricular systolic beat. A phonocardiogram showed no murmurs; however, a gallop was recorded 0.10 second after the second sound just below the xiphoid area, the characteristic protodiastolic sound of constrictive pericarditis. The jugular pulse had a rapid and deep V-wave. Roentgenkymograms demonstrated a rapid filling of the ventricle with a diastolic plateau (Fig. 2).

Fig. 1.—The posteroanterior roentgenogram showing the abnormal cardiac silhouette.

Pressure curves characteristic of constrictive pericarditis with right ventricular "diastolic dip" and plateau with end diastolic pressure above the normal range were noted on cardiac catheterization. Following exercise, the right ventricular systolic pressure did not rise significantly but there was a definite increase in the end diastolic pressure of the right ventricle.

TABLE I. THE PRESSURES RECORDED AT THE TIME OF CARDIAC CATHETERIZATION

| | VOL. PER CENT $O_2$ CONTENT | MM. Hg PRESSURE |
|---|---|---|
| "P.C." | — | 9 |
| P.A. | 9.9 | mean 19: 31/14 |
| R.V. | — | 31/11 |
| R.A. | — | mean 11 |

superior vena cava with the right atrium, there was a definite firm constricting fibrous band of pericardium which was dissected in order to relieve the obstruction of the superior vena cava. It was this band which was described preoperatively in the angiocardiographic study. The patient has continued to show slow but progressive improvement since her discharge on Sept. 6, 1955.

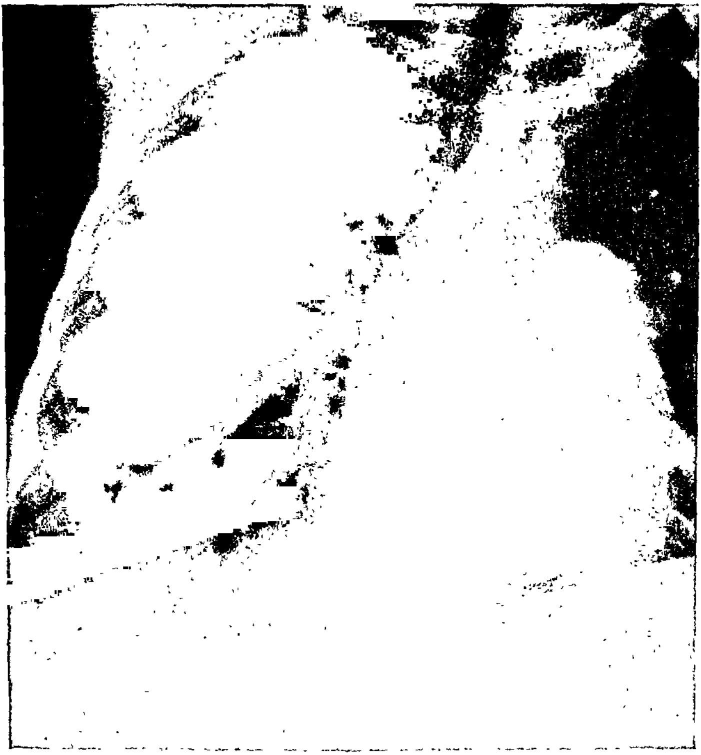


Fig. 4.—The five-second angiocardiogram showing the enlarged right atrium, the right ventricle, and pulmonary circulation opacified. One can see the flecks of calcium present and also note the opacification of a dilated inferior vena cava.

## DISCUSSION

This report constitutes the first preoperative recognition of a constriction of the superior vena cava associated with constrictive pericarditis and demonstrated by angiocardiography. Steinberg[3] has not had this experience. This finding was proved by operation. It is most important to note this finding in patients with constrictive pericarditis, for the reasons Holman[1, 2] has mentioned, to obtain a successful result for the operation. Although the

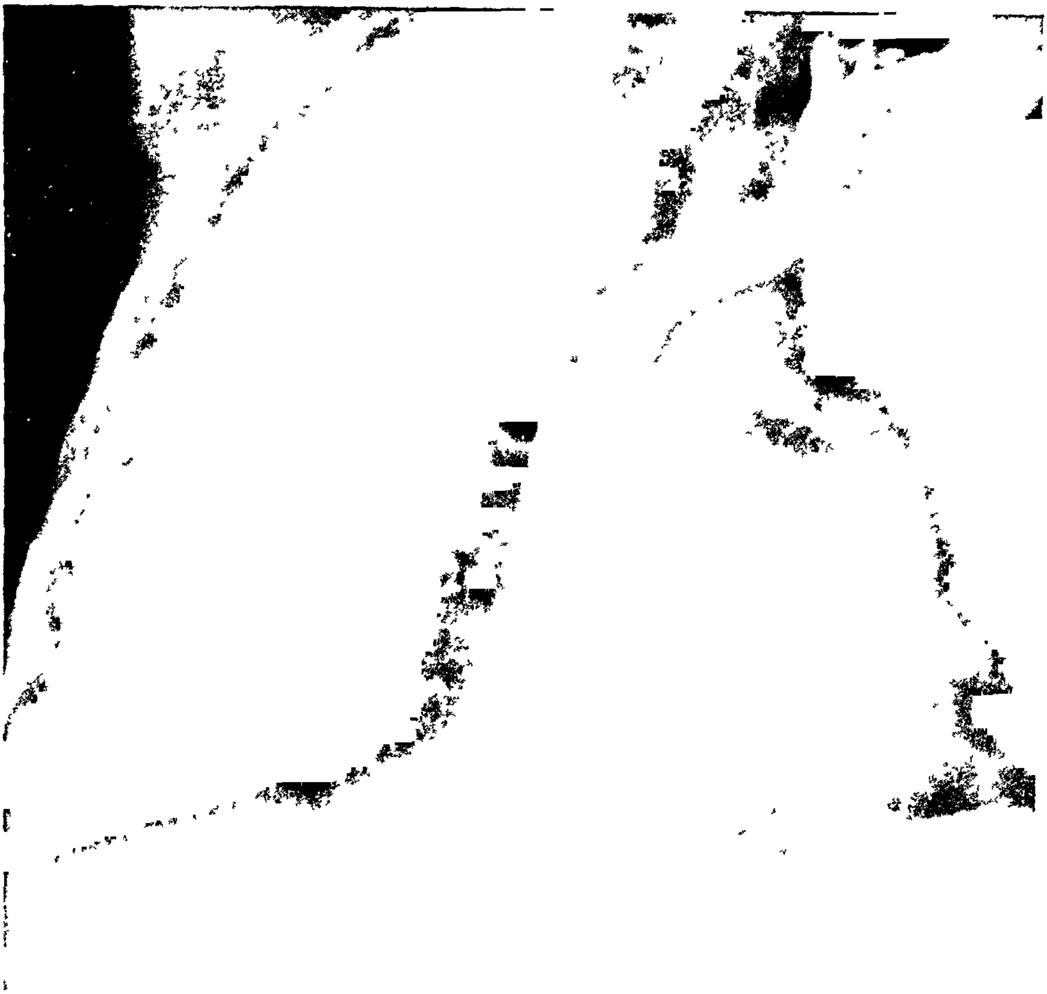


Fig. 3A.—The first-second angiocardiographic film showing the band of constriction around the superior vena cava at its junction with the right artium as these are opacified.

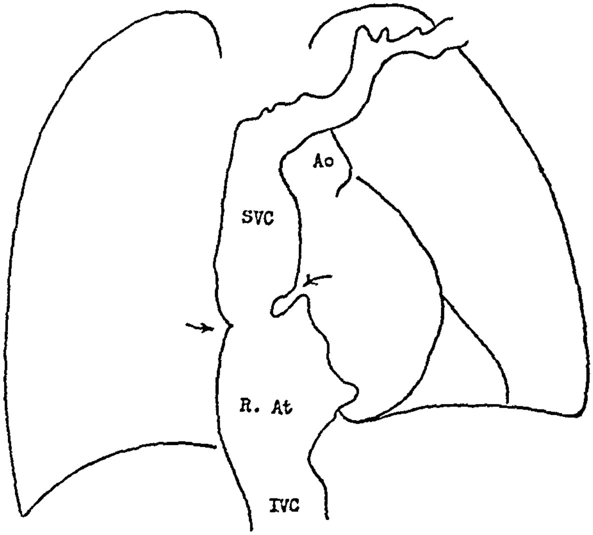


Fig. 3B.—A drawing demonstrating the area of superior vena caval constriction.

# CONGENITAL ABSENCE OF THE AORTIC ISTHMUS

## Report of a Case With Successful Surgical Repair

Duane L. Merrill, M.D.,* Charles A. Webster, M.D.,** and
Paul C. Samson, M.D.***
Oakland, Calif.

ONE of the rarest anomalies of the aortic arch is complete absence of the isthmus, that portion of the arch between the origins of the left subclavian artery and of the ductus arteriosus. Functionally, this abnormality is the most extreme form of juvenile or preductal coarctation. The ductus arteriosus persists, continues on as the descending aorta, and is referred to by Evans[13] as the "pulmonary-ductus-descending aorta trunk."

In a study of 200 cases of adult coarctation, Maude Abbott[2] found a persistent patent ductus arteriosus entering the aorta at, or proximal to, the coarctation in 10 per cent. Baffes[4] recently reviewed 105 operative and autopsied cases of coarctation of the aorta of all types and found atresia of the descending arch in 2 cases (1.9 per cent). In none was the isthmus completely absent. In a review of 19,217 routine autopsies, Evans found an incidence of all varieties of coarctation in 28 cases (0.13 per cent). Complete interruption of the aortic arch with no demonstrable connection between the arch and the pulmonary-ductus-descending aorta trunk was found in three instances In one of these (Case 22) a patent foremen ovale was present and the great vessel abnormalities were anatomically identical with the case to be described. Anomalous origins of the subclavian arteries from the descending aorta were found in the other 2 cases; one had a patent interventricular septal defect. In 1948, Sewart[16] reported one case of congenital interruption of the aortic arch and made reference to 6 previously reported cases, 5 collected by Abbott,[1] and the one found by Evans. Sewart's case had a high interventricular septal defect; in none of the others was such a defect reported.

This report concerns a 3½-year-old white girl in whom this anomaly was found at operation and definitive repair of the lesion accomplished. To our knowledge, this is the first instance in which the lesion has been encountered in a living child and successfully corrected surgically.

### CASE REPORT

P. R. (S. M. H #5962U) was admitted to the Samuel Merritt Hospital by Dr. Karl Kennedy for study and observation on June 12, 1955. Her age was 3½ years and her

---

From the Cardio-Respiratory Center of the East Bay, and the Thoracic Surgery Service, The Samuel Merritt Hospital Oakland, Calif

Received for publication Feb 3, 1956

*Assistant Resident in Thoracic Surgery, Highland-Alameda County Hospital and The Samuel Merritt Hospital, Oakland, Calif

**Director, The Cardio-Respiratory Center of the East Bay, Oakland, Calif.

***Chief of Surgery The Samuel Merritt Hospital, Oakland, Calif.

catheterization pressure studies did not indicate an obstruction at the atrial inlet, the operative findings did corroborate the angiocardiographic evidence. This was considered to be of significance. It is this experience which points to the need for the use of angiocardiography as an additional preoperative diagnostic method. Since the maximal result of pericardiectomy may not be realized should such defects remain uncorrected by surgery, it is best to try and demonstrate them before operation.

## REFERENCES

1. Holman, E., and Willett, F.: Results of Radical Pericardiectomy for Constrictive Pericarditis, J. A. M. A. 157: 789-794, 1955.
2. Holman, E., and Willett, F.: The Surgical Correction of Constrictive Pericarditis, Surg., Gynec. & Obst. 89: 129-144, 1949.
3. Steinberg, I.: Personal communication.
4. Holman, E.: Personal communication.
5. Delorme, E.: Sur un traitment chirurgical de la symphyse cardiopericardique, Bull. et Mém. Soc. chir. de Paris 24: 918, 1898.
6. Churchill, E. D.: Decortication of the Heart (Delorme) for Adhesive Pericarditis, Arch. Surg. 19: 1457, 1929.

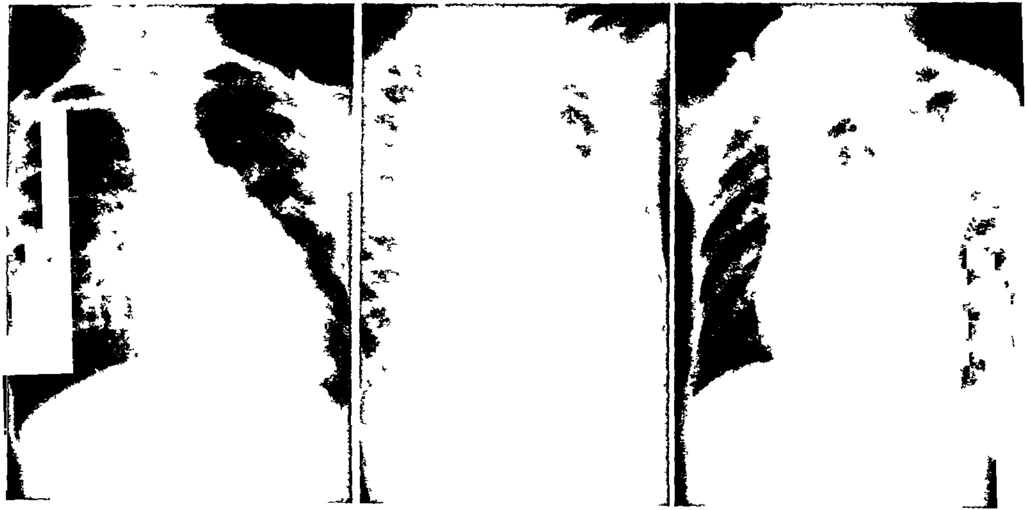


Fig 2—Preoperative roentgenograms of the chest in posteroanterior right anterior oblique and the left anterior oblique projections There is an over all increase of the size of the heart with obvious hypertrophy of both left ventricle and right ventricle There is considerable fullness of the right ventricular outflow tract and the hilar shadows of the pulmonary arteries are prominent It is difficult to distinguish an aortic knob There is no evidence of auricular enlargement

weight, 28 pounds; obviously a delay in growth. A heart murmur had been present since birth. The mother's pregnancy had been normal full term. The birth weight was 7 pounds. The child was normally active, but had always been a feeding problem. There were occasional respiratory infections complicated by mild asthma. Cyanosis had never been noticed, nor had there been evidence of cardiac failure.

Physical examination revealed a thin, underdeveloped girl with moderate pectus carinatum. The blood pressure in the right arm was 160/80 mm. Hg and in the right leg 125/80 mm. Hg. The heart was enlarged with the apex in the sixth left intercostal space in the anterior axillary line. The apical pulsation was readily palpable but without precordial heave or thrill. There was a loud, harsh, low-pitched systolic murmur with maximum intensity in the fourth and fifth intercostal spaces 2 cm. to the left of the sternum; this murmur was widely transmitted over the precordium. There was a rumbling diastolic murmur at the apex. A ntinuous murmur was heard in the left infraclavicular region with a soft diastolic comment, but this could not be differentiated from the murmur heard lower in the chest. The ılmonic second sound was accentuated and split. The lung fields were clear. There was no ubbing of the digits; the color of the fingers and toes was compared and no visible difference ›ted. The examination was otherwise normal throughout.

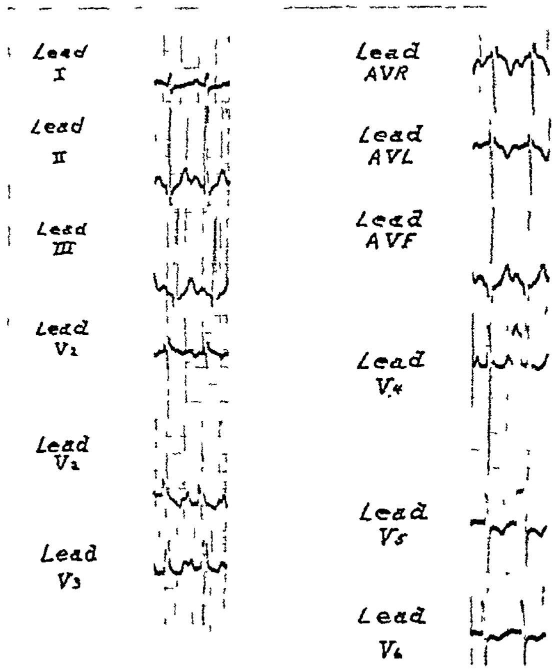


Fig. 1.—The preoperative electrocardiogram made on April 18, 1955.

Laboratory studies showed the hemoglobin to be 13.6 Gm.; red blood count, 4.59 million; white blood count, 5,700 with a normal differential. An electrocardiogram showed partial right bundle branch block and inversion of T-waves over the entire precordium. This was interpreted as presumptive evidence of right and left ventricular hypertrophy (Fig. 1). Preoperative roentgenograms of the chest are shown in Fig. 2.

Cardiac catheterization was performed on June 13, 1955, by one of us (C. A. W.) under Demerol and Pentothal anesthesia. During catheterization visible cyanosis of the toes was noted, while the fingers remained normally pink. The results of catheterization are shown in Table I.

These data were interpreted as showing, distal to the origin of the left subclavian artery, a right-to-left shunt such as is seen in a "reverse flow" type of patent ductus. An left lung and continued as an even larger descending aorta, this area corresponding to the usual location of a patent ductus arteriosus The isthmus of the aorta between the left subclavian artery and the insertion of the ductus was absent The recurrent laryngeal branch of the vagus nerve was identified as it hooked around the area of the "ductus", it was then followed proximally where it appeared to lie posterior to the enlarged and tortuous subclavian artery No thrill could be palpated proximally between the aorta and the pulmonary artery, therefore, we concluded there was no aorticopulmonary artery septal defect (Fig 3)

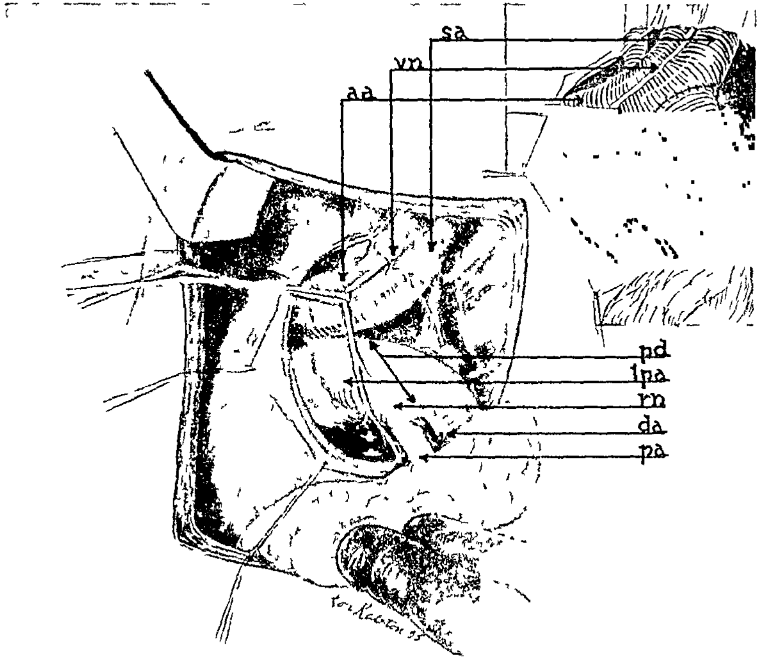


Fig 3—High left posterolateral incision Drawing at operation Inset shows method of repair *sa*, Left subclavian artery *vn*, left vagus nerve *aa*, aortic arch *lpa*, left main pulmonary artery *rn*, left recurrent nerve *da*, descending aorta *pd* region of patent ductus at its juncture with pulmonary artery, *pa*, branch of left main pulmonary artery to left lung

Pressures recorded in the main and left pulmonary arteries were 70/37 mm Hg, indicating that there was no stenosis between these vessels The pressure in the descending aorta was 78/45 mm Hg When the vessel, corresponding to the "ductus," was clamped the pressure in the descending aorta rose to 125/104 mm Hg and that in the pulmonary artery decreased to 63/22 mm Hg; the aorta became obviously more distended, while the pulmonary artery became less so This clearly demonstrated that there was a significant blood flow, under relatively higher pressure, through collateral vessels from the ascending aorta and branches, to the descending aorta Physiologically, the pressure changes were similar to

TABLE I. DATA FROM PREOPERATIVE CATHETERIZATION

| CHAMBER | OXYGEN (VOL. %) | PRESSURE (MM. HG) |
|---|---|---|
| Superior vena cava | 12.2 | |
| Inferior vena cava | 8.8 | |
| Mid-right auricle | 11.6 | 3 (mean) |
| Tricuspid right ventricle | 11.5 | |
| Mid-right ventricle | 12.1 | 83/18 |
| Pulmonic right ventricle | 12.1 | |
| Main pulmonary artery | 12.1 | 89/75 |
| Left pulmonary artery | 12.1 | 95/74 |
| Right femoral artery | 13.8 | 95/53 |
| Right brachial artery | 15.3 | 120/66 |
| *Repeat* | | |
| Left pulmonary artery | 11.6 | |
| Main pulmonary artery | 11.8 | |
| Pulmonic right ventricle | 10.7 | |
| *Arterial Oxygen Saturation* | | |
| Femoral artery | 84.0% | |
| Brachial artery | 93.0% | |

incomplete preductal coarctation seemed the most likely explanation for the pressure differential between brachial and femoral arteries; it was thought that this might also explain the relatively mild femoral arterial oxygen unsaturation in spite of the ductus-to-aorta shunt. A ventricular septal defect was suspected because of the heart murmur and the slight increase in oxygen content of the right ventricle over the right auricle.

The possibility of complete preductal coarctation with persistent ductus was discussed, but in the absence of clinically demonstrable collateral flow we could not adequately explain the relatively good saturation of the femoral artery blood.

On June 27, 1955, exploratory thoracotomy was performed through a left posterolateral approach (P. C. S.). The anesthesia was Pentothal and nitrous oxide with oxygen. With the initial incision it became apparent at once that large anastomotic channels were present even in the subcutaneous tissues. The evidence of an extensive collateral circulation continued as the muscles were divided and the fifth rib removed. Upon entering the pleural cavity, the posterior portion of the left hilum was examined and we were then first aware of fiercely pulsating vessels extending up into the neck and in the internal mammary region. There was a large, but relatively quiet, descending aorta. Palpation of the pulmonary artery showed it considerably more tense than normal and greatly enlarged. The aortic arch, left common carotid, and left subclavian vessels were dissected free and the enlarged collateral intercostals were likewise isolated and preserved. The arch seemed to *end* in the subclavian artery. These vessels as well as an innominate branch seemed to have normal distributions peripherally as tested by pulse obliteration techniques. The vagus and recurrent laryngeal nerves were identified and gently retracted medially.

The pericardium was entered just posterior to the phrenic nerve and the enormous pulmonary artery isolated. There was a diastolic thrill felt in the pulmonary artery and a systolic thrill felt posteriorly on the heart, most marked at the junction of the left auricle and ventricle. This was short and fairly sharp.* No thrills were palpated either at the base of the heart or at the apex. The pericardial incision was carried out along the left pulmonary artery which seemed to empty into the descending aorta; however, no coarctate area could be found.

When the entire anatomy was worked out it was seen that the main pulmonary artery first divided normally; then the left main pulmonary artery gave off a large branch to the patient was rapidly given 200 c c of whole blood and placed in Trendelenburg position, there was an immediate return of the blood pressure to a systolic mean of 120 By this time the pulmonary artery pressure had fallen to 52/37 mm Hg The pericardium was closed loosely over the suture line in the pulmonary artery The aortic anastomosis was covered with Gelfoam and reinforced with pleura A small lung biopsy was taken The chest was closed in layers with silk One right angle intercostal catheter was placed under water seal, the patient returned to the Recovery Room in good condition In the immediate postoperative period she required direct laryngoscopy and tracheal aspiration on one occasion

---

*Subsequent observations, in a patient with the adult or postductal type of coarctation in which the collateral circulation was extensively developed, demonstrated a similar thrill at the atrioventricular junction which could be obliterated by gentle compression of the left circumflex coronary artery. This thrill was attributed to increased flow and tortuosity of the coronary artery which seemed to participate in the generalized collateral development. The possibility remains in the present case, however, that the thrill at this point may be due to a high interventricular septal defect as suggested by the catheterization studies.

For the first two weeks the clinical course was disappointing There were persistent low grade fever (101°, rectally), leukocytosis, anorexia, slight weight loss, a fall in hemoglobin, and progressive irritability and lethargy She remained fully digitalized and there was no evidence of cardiac failure Early adventitious pericardial sounds developed into a full blown pericardial friction rub on and after the sixth postoperative day Chest x rays fifteen days postoperatively (Fig 4) showed an increase in the heart shadow suggesting pericardial fluid The rapid development of neck vein distention with pulsation and hepatomegaly suggested tamponade A pericardial tap yielded 40 c c of bloody fluid

On the seventeenth postoperative day, anterior pericardiotomy and transpleural drainage of the pericardial sac were done by resecting the left fifth cartilage and a portion of the rib Approximately 150 c c of old bloody fluid and a considerable amount of fibrin were removed from the pericardium

There was an early fibrinoplastic peel over the epicardium, but it did not seem advisable to decorticate the heart There was, likewise, an early angioblastic and fibroblastic peel of 1 or 2 mm thickness on the pericardial side which was removed in part The pericardium was closed anteriorly but widely and dependently drained by a cruciate incision posterior to the left phrenic nerve This should have been performed at the original operation instead of merely relying on the loose anterior closure of the pericardium for adequate drainage Smears and cultures of the removed peel and fluid were negative for pyogenic organisms The pericardial friction rub rapidly disappeared and on fluoroscopy the heart appeared much smaller Thereafter her hospital course was that of gradual improvement, and she was discharged July 25, 1955

During the intervening seven months the patient has gained 2½ pounds in weight and ¾ inch in height The blood pressure in the legs is 130/84 mm Hg She is considerably more active and alert, she tires much less readily Digoxin was discontinued after six months, and cardiac compensation has been well maintained The patient has had a single respiratory infection with bronchitis There has been little change in the heart size since operation, but there is a decrease in the region of the pulmonary artery, and the hilar vascular markings have lessened There is a persistent harsh murmur to the left of the sternum in the fourth interspace, suggesting the presence of a ventricular septal defect

## DISCUSSION

Complete interruption of the arch of the aorta is an extremely rare disorder The persisting circulatory pattern is fetal in type The absent segment of arch may lie between the left common carotid or subclavian arteries, and the descending aorta at the juncture of the ductus arteriosus This segment develops embryologically from the fourth aortic arch and the third to seventh dorsal aortic segments[3, 9, 11] The process of involution or maldevelopment can only be surmised, but it poses interesting considerations The subclavian vessel normally develops as a branch of the seventh dorsal intersegmental artery caudad to the sixth aortic arch (which becomes the ductus) During early embryonic growth as the heart descends, the left subclavian shifts cephalad to the ductus or sixth arch, and comes to lie in its normal relation to the left common carotid artery In the case just reported, the portion of arch between the left subclavian artery, those found in patients with patent ductus arteriosus with pulmonary hypertension, intermittent reversal of flow, and predominant left-to-right shunt. They indicated that the "ductus" could and should be divided to reduce pulmonary tension and yet assure adequate flow to the caudal portion of the body.

Attention was then given as to how we could best attack the lack of isthmus and it seemed quite logical that an anastomosis should be made between the arch of the aorta and the descending aorta, which once divided from the pulmonary artery, could be brought up and anastomosed to the base of the left subclavian artery; therefore, the aortic homogratt which we had prepared did not seem necessary.

Fig. 4.—Posteroanterior roentgenogram of chest made fifteen days postoperative There extensive bilateral pulmonary congestion The cardiac shadow is grossly enlarged as compared with preoperative studies and is globular in shape These changes may well be due to increased pericardial fluid with impending tamponade

By using clamps across the "ductus" at the pulmonary artery and below on the descending aorta, the ductus was severed and the pulmonary artery was then closed with a continuous mattress and return over and over suture of No. 5 0 Deknatel silk. The edge of the cut aorta was beveled slightly in the other direction to give a total inside diameter of 12 mm. A medium sized auricular appendage clamp was placed across the junction of the subclavian and the left common carotid vessels and the subclavian artery was then completely occluded by a bull dog clamp. Care was taken not to obliterate the left temporal pulse with the proximal clamp. A 12 mm. longitudinal incision was made at the base of the subclavian artery which extended up the lateral arterial wall for a short distance. The end-to side anastomosis was completed using continuous over and over No. 5 0 Deknatel silk, interrupted twice (Fig. 3, inset). Following slow removal of all the clamps there was a good pulsatile expansion of the descending aorta which had not been present before. During these maneuvers there was a moderate fall in systolic blood pressure to 80 mm. Hg. The prenatal development without collateral channels Following birth, the blood supply to the distal aorta continued through the ductus and the interventricular shunt became a source of oxygenated blood to the trunk and legs

Three factors may have contributed to the development of the extensive arterial collateral bed demonstrated at operation (A) oxygenated blood flowing left to right through the septal defect and via the ductus to the descending aorta may have allowed sufficient *time* to elapse for the development of collaterals, (B) continued arterial unsaturation (chronic tissue anoxia) may well have acted as a stimulus to collateral formation within a year or two, and (C) following birth, persistent hypertension in the arch and branches of the proximal aorta may have stimulated existing but undeveloped collateral channels to dilate and open[15] The finding in adult coarctation that the coronary arterial system shares in generalized tortuosity and dilatation with other branches of the proximal aorta is offered in support of this theory

Repair of the lesion in the manner described seemed most satisfactory Because the exact lesion was unsuspected prior to thoracotomy the technical details were decided at operation without recourse to suggestions appearing since, in the literature Bailey did describe the utilization of the divided ductus to anastomose to the proximal aorta in cases of preductal coarctation and suggested the repair of absence of the isthmus in a similar fashion Gerbode and associates[14] have reported the case of a patient with preductal coarctation with a large patent ductus arteriosus and an intracardiac shunt who was operated upon at the age of three months No enlarged collateral vessels were seen It was possible to resect the coarctation from the base of the left subclavian artery to just beyond the ductus, ligate the ductus at the pulmonary artery, and mobilize the aorta sufficiently to perform an end to end anastomosis On experimental grounds, Baffes has suggested the use of aortic homografts from the ascending to the descending aorta in preductal coarctation and in atresia of the isthmus

Although there was minimal narrowing of the newly formed isthmus in the present case, this was not disturbing A slight narrowing of the isthmus is a normal finding during the first three months of postnatal life in 80 per cent of infants with normal hearts[18] and may persist into adult life in many instances We know that aortic flow has been improved and that the pulmonary hypertension has been relieved insofar as the extracardiac circulation is concerned Whether or not the probable interventricular septal defect will prove now to be a hazard remains to be seen

## SUMMARY

A report is made of the first successful operative correction of an extremely rare congenital abnormality of the aortic arch In this case, the isthmus of the aorta was absent and the descending aorta arose from the left main pulmonary artery via a large patent ductus arteriosus There was an unusually large collateral blood flow from branches of the aortic arch to the descending aorta and a bidirectional but predominantly left to right shunt through the ductus The large collateral flow explained the clinically misleading findings of palpable femoral pulses and lack of cyanosis of the toes

and the ductus was absent which would indicate an involution at a later embryonic stage after the subclavian had shifted cephalad. In 2 cases reported by Evans, however, the left subclavian arose from the descending aorta, and the absent segment extended from the internal carotid to the ductus-aortic juncture. This would suggest involution at an earlier stage of embryonic development.

Complete interruption of the arch is ordinarily incompatible with a duration of life longer than early infancy because of absent or poor collateral at birth; in fact, Bailey[7] has stated that collateral vessels are *not* present in absence of the isthmus. In this and in other respects, the lesion, functionally, is the most extreme form of juvenile or preductal coarctation. Occasionally, however, one sees an adult with the infantile type, or an infant with the adult-type of coarctation; this has prompted Bahnson[6] to suggest that, surgically speaking, coarctation be designated as complicated or uncomplicated.

In the patient under discussion and in others in whom there is an interruption in oxygenated blood supply to the descending aorta, it is the development of adequate collateral flow which sustains life. Bahn and his colleagues[5] have discussed coarctation of the aorta as a cause of death in early infancy. They presented three autopsy cases in which the ductal flow was into the descending aorta. The collateral channels in each case were poorly developed, and they concluded that death resulted from left ventricular failure secondary to hypertension. Edwards[12] believes that one reason for the extreme rarity of absent isthmus is that death usually may be attributed to normal rapid closure of the ductus in the immediate postnatal period with resultant inadequate flow to the descending aorta. Ballantyne[8] explained the difference in the course and prognosis of patients with the adult and the infantile types of coarctation by stating that in the adult type in which there is an obstruction to the aortic flow at or below the ductus, it is necessary for collateral channels to be established early in embryonal life if the fetus is to survive in utero. In the preductal type (and in absence or atresia of the isthmus), adequate circulation in the distal aorta is maintained via the ductus and no stimulus to the establishment of collateral circulation exists until after birth. Usually a collateral circulation does not develop rapidly enough to sustain life.[10, 17]

In the case described by Sewart the child lived for nearly forty-six hours. During this time it was noted that there was no cyanosis in the lower extremities. In view of the autopsy findings, it was suggested that sufficient oxygenated blood was being shunted into the pulmonary circulation through the interventricular septal defect, which was found to mask any clinical desaturation in the lower half of the body. In the 5 cases presented by Abbott,[1] and in the one described by Evans, no interventricular septal defect was reported. All but one of these patients died in infancy, the exception living to the age of 5½ years. In Abbott's cases, the ascending aorta was thin and hypoplastic. The descending aorta was in each instance a continuation of a widely patent ductus arteriosus; all patients had generalized cyanosis. Abbott explained this by assuming a reverse flow through the collateral bed from lower to upper aorta.

An intracardiac shunt may well explain the survival of our patient past infancy. With a large patent ductus, fetal circulation was adequate for full

# PECTUS EXCAVATUM

## SURGICAL TECHNIQUE A NEW FORM OF EXTERNAL TRACTION TO THE ELEVATED STERNUM

C LEON HOLMES, M D
JACKSON, TENN

PECTUS excavatum, or funnel chest is a congenital, sometimes familial deformity affecting the sternum and adjoining cartilages The etiology is not clearly understood though the most commonly advanced theory is that first suggested by Woillez[13] in 1860 He postulated the presence of a short central tendinous portion of the diaphragm which held the xiphisternal junction in a depressed state that became more pronounced as growth occurred This theory was strongly seconded by Brown,[3] Oschner and DeBakey,[9] Sweet,[17] and Lester Brodkin,[2] however, suggested that the deformity was due to a deficient muscular development of the anterior portion of the diaphragm which is derived from the septum transversum Whatever the etiology, the deformity is psychologically distressing *to the parents, and later to the child It is, more importantly, a* severe physical defect that in many instances leads to real disability, both respiratory and cardiovascular

### INDICATIONS FOR TREATMENT

Until recent years treatment was undertaken only rarely The first reported instance was that of Ludwig Meyer[8] which was carried out in 1911 In 1913, Sauerbruch[11] reported his first case and he later added other cases to the literature using slightly different techniques in each one reported Following these pioneer procedures, sporadic reports appeared with increasing frequency but up until the time of Oschner and DeBakey's comprehensive review[9] in 1938, only 32 operative cases had been reported Following this study and that of Brown,[3] a much greater interest in the surgical management of this condition became generally manifest It is now generally agreed that correction is indicated, (1) to correct the sternal depression, (2) to prevent, or partially correct the anteroposterior flattening of the entire chest, and (3) to relieve dyspnea, pulmonary complications, exercise intolerance, cardiac arrythmias, murmurs, or other evidences of cardiopulmonary limitation

Others have added to these indications, the need for early correction to prevent psychologic disturbances in the affected child All are aware of the forthright frankness of children which can be disarming in its simplicity on one occasion while utterly devastating on another One can only imagine which effect childish observations of this deformity would have on an innocent victim It is the wonderful virtue of adaptation that leads most of us through emotional

---

Received for publication Feb 7 1956

The embryology, physiopathology, and surgical repair of this lesion are discussed.

REFERENCES

1. Abbott, M. E.: In Osler and McCrae: Modern Medicine, London 4: 772, 1927.
2. Abbott, M. E.: Coarctation of the Aorta of the Adult Type, II. A Statistical Study and Historical Retrospect of 200 Recorded Cases, With Autopsy, of Stenosis or Obliteration of the Descending Arch in Subjects Above the Age of Two Years, Am. Heart J. 3: 574, 1928.
3. Arey, L. B.: Developmental Anatomy, Philadelphia and London, 1940, ed. 4, W. B. Saunders Company.
4. Baffes, T. G.: Adaptation of Homologous Aortic Grafts for Surgical Correction of Infantile Coarctation of the Aorta, Surgery 38: 486, 1955.
5. Bahn, R. C., Edwards, J. D., and DuShane, J.: Coarctation of the Aorta as a Cause of Death in Early Infancy, Pediatrics 8: 192, 1951.
6. Bahnson, H. T.: Coarctation of the Aorta and Anomalies of the Aortic Arch, S. Clin. North America 32: 1313, 1952.
7. Bailey, C. P.: Surgery of the Heart, Philadelphia, 1955, Lea & Febiger.
8. Ballantyne, E. N.: Coarctation of the Aorta (Report of 3 Cases), Am. J. Dis. Child 50: 642, 1935.
9. Barry, A.: The Aortic Arch Derivatives in the Human Adult, Anat. Rec. 111: 221, 1951.
10. Caldoney, M. M., and Carson, M. J.: Coarctation of the Aorta in Early Infancy, J. Pediat. 37: 46, 1950.
11. Congdon, E. D.: Transformation of the Aortic Arch During the Development of the Human Embryo, Carnegie Contributions to Embryology 277: 47, 1922.
12. Edwards, J. E.: In Gould, S. E.: Pathology of the Heart, Springfield, 1953, Charles C Thomas, Publisher.
13. Evans, W.: Congenital Stenosis Coarctation, Atresia, and Interruption of the Aortic Arch (A Study of Twenty-eight Cases), Quart. J. Med. 26: 1, 1933.
14. Gerbode, F., Purdy, A., Alway, R. H., Piel, J. J., and Da Costa, I. A.: Surgical Treatment of Coarctation of the Aorta in Infancy, Am. J. Surg. 89: 1138, 1955.
15. Quiring, D. P.: Collateral Circulation, Philadelphia, 1949, Lea & Febiger.
16. Sewart, M.: Congenital Interruption of the Aortic Arch, Arch. Dis. Childhood 23: 63, 1948.
17. Taussig, H. B.: Congenital Malformations of the Heart, New York, 1947, Commonwealth Fund and Harvard University Press.
18. Theremin, E.: Études sur les affections congenitales du coeur, Paris, 1895.

they may be, cannot be compared with the more general benefits inherent in the operation at an earlier age. Elevation of the sternum in adults, for instance, can do nothing to correct the marked flattening of the chest which has developed and become fixed through the earlier years, therefore greatly augmented pulmonary reserve cannot be promised.

At the other extreme is the very young infant. It has been stated repeatedly that surgery performed on a child under six months of age or so will be corrective if division of the posterior diaphragmatic attachments only is carried out, while more radical surgery will be necessary at a later date, even a few months later. I am skeptical of this idea. The more marked deformities might be treated by surgery in the first few months with improvement, but it is questionable if the advantages of a perhaps simpler operation are great enough to offset the disadvantages of this type of surgery at such a young age. Most surgeons prefer to delay all but emergency procedures to a time when the child is a better surgical risk, when all phases of his care are more easily and safely handled.

There are exceptions to all rules, but generally speaking, it is felt that 3 to 5 years is the optimal age for the repair of pectus deformities. Moreover, many borderline cases in the first few months of life will, in 2 to 3 years, have completely disappeared or will have developed to such a state that they can be selected for surgery without doubt as to the need for such surgery.

## SURGICAL TECHNIQUE

The review of Oschner and DeBakey covers most of the techniques employed in this condition. Since that time Brown,[3] Adams,[1] Dailey,[4] Lester,[7] Dorner,[5] and others have added details or modified techniques to improve the results achieved. Most of these techniques and their modifications have been concerned with the form of fixation of the sternum. One may wedge the deformed cartilages at their insertion or articulation with the sternum, and again at the costochondral juncture, and after elevation of the sternum these ribs and cartilages are reconstituted by suture after the method of Gross[6] (Fig. 3). One may or may not add some form of external traction to this technique. Gross does not feel it is necessary.

Others, and I agree, prefer the technique of Ravitch,[10] in which the deformed cartilages are resected and discarded, preserving the perichondrium and the attachments of this and the intercostal bundles. To maintain the elevation of the sternum, which should actually constitute an overcorrection, some form of support is necessary for a period of time. Adams, Lester, Dailey, Dorner, and others have used a rib strut, slung under the elevated sternum. Generally, this is a method which has been used in adults.

A majority of operators have depended on some form of external traction such as a wire loop under the sternum, which is brought out through needle wounds lateral to the midline incision, and attached to a metal arch or other rigid support. This is maintained for 2 to 3 weeks while enough tissue fixation takes place so that the deformity will not recur but, just as important, will prevent paradoxical respiratory effort in the unstable anterior chest wall.

stresses of varying types and severity, yet some succumb to these storms, and emerge less well balanced than their associates. Certainly, emotionally crippled individuals have been observed whose affliction was due, ostensibly at least, to this marked deformity, so it may be a point well taken to consider the emotional trauma to which a child with a marked pectus deformity is subjected, but there are such real and tangible physical reasons for advising correction, that we should not dwell too long on the potential psychic aspects (Figs. 1 and 2).

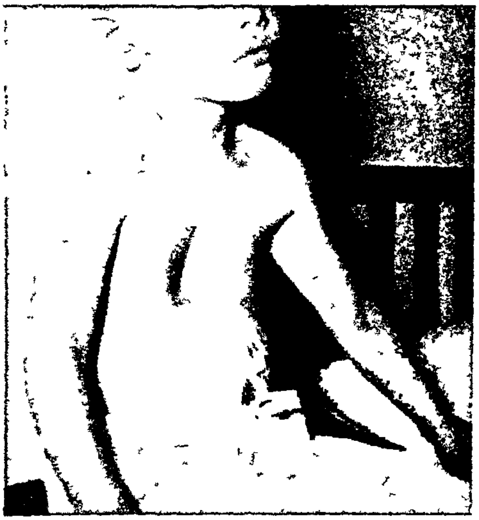


Fig. 1.

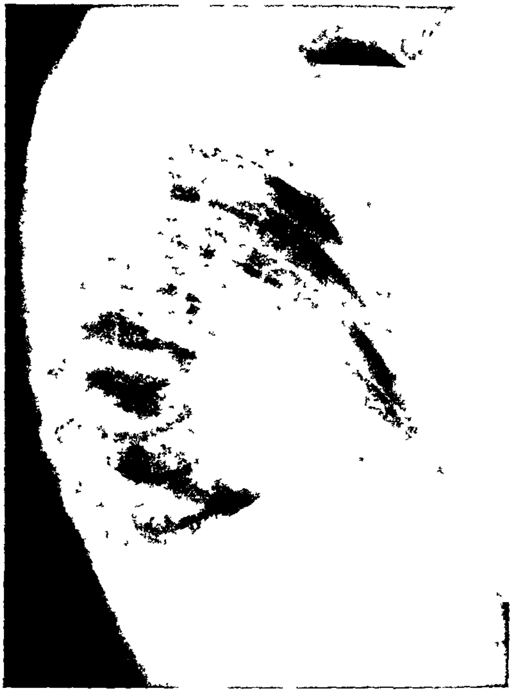


Fig. 2.

Fig. 1.—Preoperative photograph showing typical deformity in pectus excavatum.

Fig. 2.—Lateral x-ray of chest. This shows the sternal depression better than any other x-ray projection.

With respect to the age at which such treatment should be carried out, there is no universal rule. There is no age from infancy to middle age when at least some of the undesirable features of pectus excavatum cannot be alleviated with considerable benefit. Yet one should have a fairly well-defined objective when advising the operation in adults as the benefits are limited in comparison to those inherent if the operation is carried out early in childhood. Elevation of the sternum in an adult may increase pulmonary function somewhat; it may alleviate cardiac compression with improvement of the circulatory dynamics; it may eliminate evidences of myocardial irritability with conduction disturbances, and thereby rehabilitate the patient for a more useful and enjoyable life. For these benefits, one must be prepared to advise and carry out surgery on adults yet it must be emphasized that the operation in adults is a formidable one, fraught with technical difficulty at the time of surgery and often beset with trying postoperative complications. Finally, such limited benefits, desirable as

All of these techniques have given good results but, as stated previously, the technique of Ravitch is preferred with some form of external traction, rather than that preferred by Gross. As a form of support in children, external traction is preferred over any fixed rib support which has the disadvantage of requiring a long accessory incision to get the rib graft and, also, because such a graft, fixed to the ribs on either side, could become a binding force as the child grew in size.

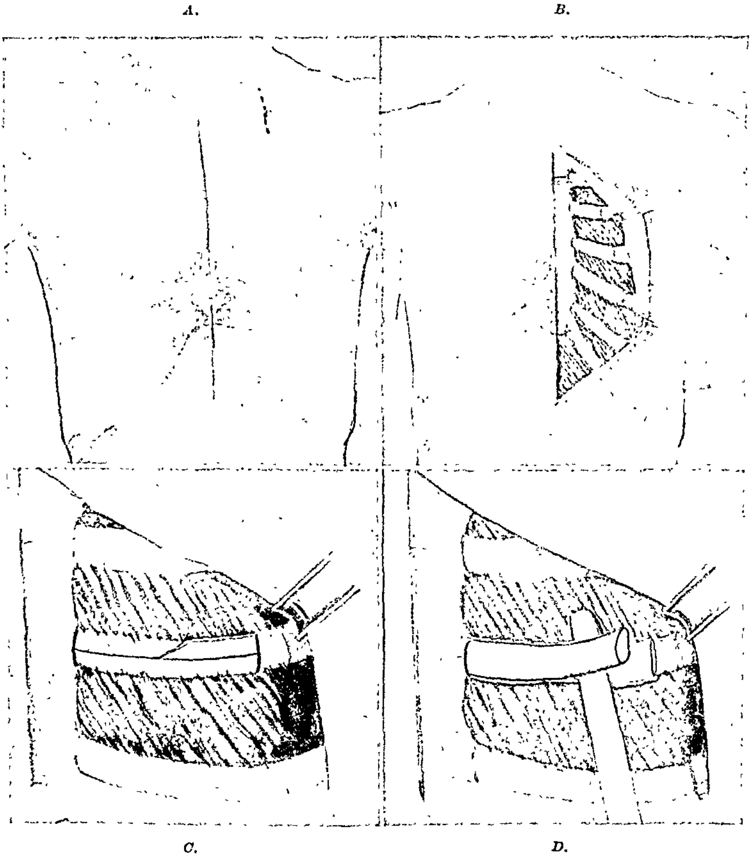


Fig. 4.—Semischematic drawings of the operative technique. *A*, Incision. *B*, Reflection of skin, subcutaneous layers, and pectoral muscles, exposing the rib cartilages and sternum. *C*, Incision in perichondrium. *D*, Peeling out deformed cartilage. (*Cont'd on next page.*)

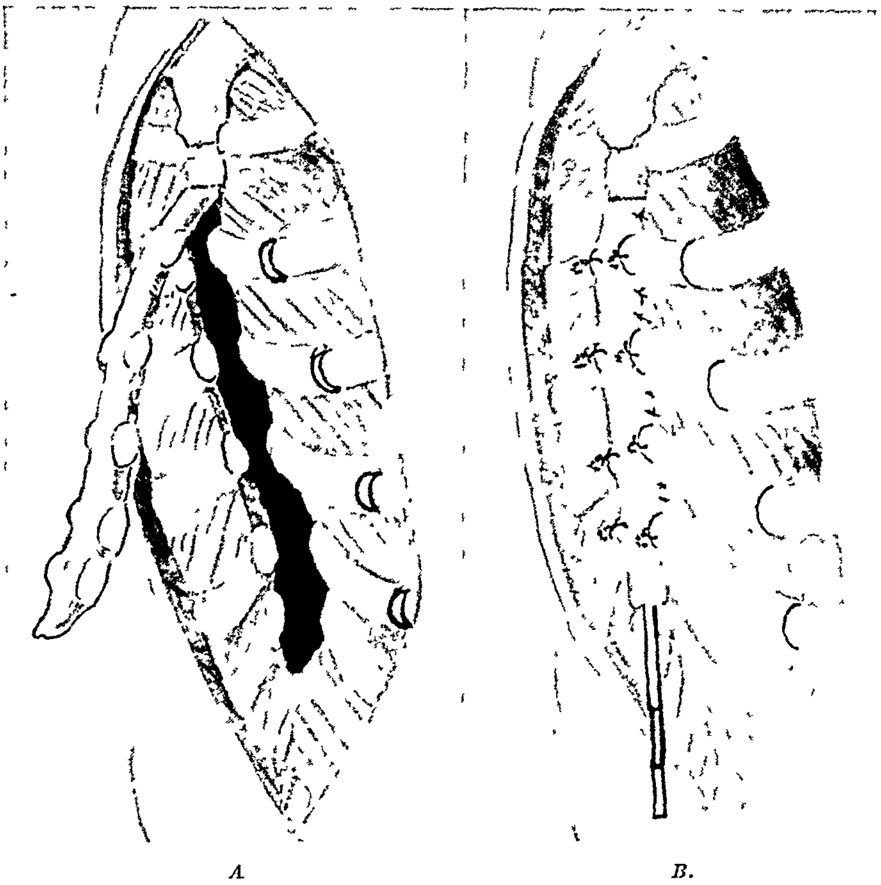


Fig. 3, *A* and *B*.—Alternate method, utilizing wedging of ribs and sternum, but no resection. *A*, Sternum free, ready for insertion of 3/32 inch pin *B*, Cartilages reattached, after wedging, with fine wire or black silk. Pin in sternum, hilt emerges from skin through separate stab wound.

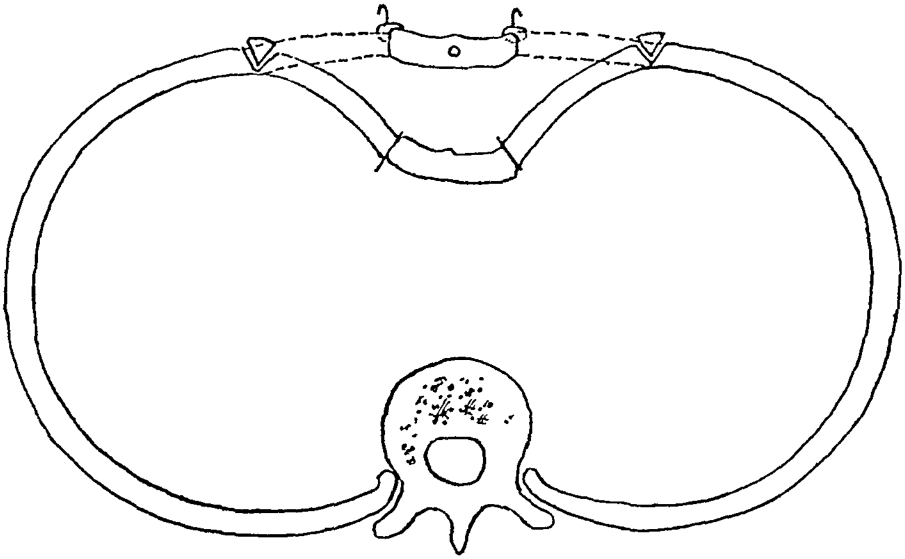


Fig 3*C*.—Cross section.

the origin of the pectorals are reflected from the sternum and the anterior ribs (Fig. 4, *A* and *B*). The perichondrium is incised and stripped from the cartilages, third to the seventh inclusive, bilaterally. These deformed cartilages are then resected at the costochondral and the chondrosternal juncture and discarded (Fig. 4, *C* and *D*). The xiphoid which is also deformed, being turned sharply forward, is elevated and resected. Any limiting diaphragmatic attachments are divided from this area of the lower sternum. With the sternum thus freed, the real degree of backward sternal curve can be observed. In all but perhaps the very youngest infants, this will be sufficient to warrant an osteotomy of the sternum at the level of the third interspace to correct the sternal depression, or to allow an adequate degree of "over correction." This osteotomy can be done in two ways. The usual method is to cut a wedge of perhaps 0.5 to 1.0 cm. through the anterior table, and bending, or actually fracturing, the posterior table to gain an adequate elevation of the sternum. Another method is to pass a Gigli saw under the sternum at the proper level and cut through the posterior cortex. This has no real advantage, though only one cut through the sternum is necessary here as the ventral bending of the sternum spreads the site of the posterior osteotomy, or saw line.

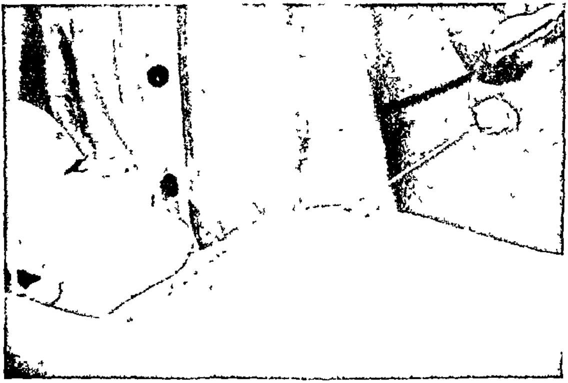


Fig 5.—Postoperative photograph. The sternum is well elevated The wire is attached to a pectoral ladder, or other scaffold, for 2 to 3 weeks.

With the sternum then completely free and held in an elevated position, a very tiny, 2 mm. stab wound is placed 1 to 2 cm. to either side of the incision at the level of the end of the sternum. Through this hole a 3/32 inch Steinmann pin is introduced with a hand drill. The pin passes through the small stab wound, and is made to engage the tip of the sternum. This pin is slowly drilled through the entire length of the medullary space, across the osteotomy site, and into the manubrium. As the osteotomy site is traversed the sternum is lined up so the pin will continue across the manubrial marrow space. It is thus that the "normal" angle is eliminated, and it is by this degree that the sternal elevation constitutes an "over correction." The drill point is stopped as it is felt to emerge from the manubrial cortex in the suprasternal notch. The hand drill used to introduce the pin is removed from the hilt.

The long operative wound is then closed with interrupted silk sutures. The instability of the anterior chest wall on either side of the sternum is negated by the elevation of the sternum This elevation in turn will be maintained by some pectoral ladder, or archlike support over the lower chest or the upper abdomen. Fig. 5 shows the pin in position and the sternum elevated after a period of traction. The wire is attached to the "ladder" which is fixed to the patient's torso. The extreme tip of the protruding pin can be bent slightly prevent the wire slipping off, losing the traction. This elevation and external traction

While external traction has been the preferred method of holding the sternum in the correct position, this too has carried with it some problems, at least on occasion. In one of my cases, the wire actually cut through the sternum which is rather soft in children, and the support was lost sooner than it was thought desirable. In other cases the wire might break. These considerations led me to act on a suggestion by an orthopedic colleague (Dr. James LeNoir of New Orleans) that a Steinmann pin be inserted through the sternum, the osteotomy site, and buried in the manubrium, the hilt being left long and the traction applied to this protruding shaft. Inasmuch as skeletal traction via Steinmann and other pins is such a well-tried and accepted procedure, this suggestion was thought worthy of trial.

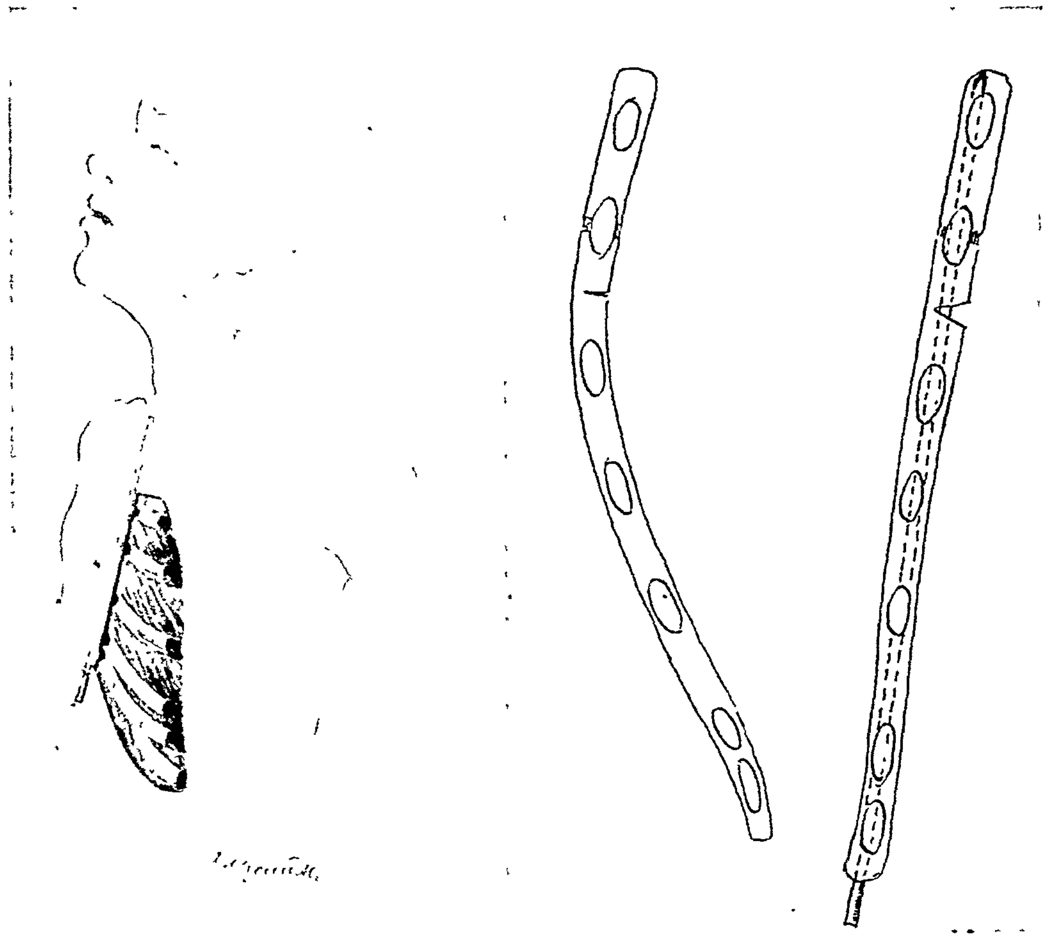

E. F.

Fig. 4.—Cont'd. *E,* Cartilages all removed, $\frac{3}{32}$ inch pin inserted through entire sternum, into manubrium. *F,* Shows $\frac{3}{32}$ inch pin (osteotomy through posterior table).

## OPERATIVE TECHNIQUE

The actual technique is shown in the photographs of a series of semischematic drawings (Fig. 4). The skin is incised in the midline, from the level of the angle of the sternum to a point midway between the xiphoid and the umbilicus. The skin, subcutaneous tissues, and to the relative infrequency with which any one surgeon will deal with the condition, (2) to the special problems imposed because of the wide mobilization of the anterior chest wall necessary to elevate the depressed sternum, and (3) to the several methods of sternal fixation introduced, internal and external

The present report introduces nothing new in the way of correcting the deformity but does report another simplified form of external traction built on the well-established principle of external traction via a Steinmann pin This method aligns the elevated and osteotomized sternum over a 3/32 inch Steinmann pin which penetrates the entire sternal marrow space and is fixed in the manubrium The free end is held suspended to a pectoral "ladder" or arch by a wire for a period of 2 to 3 weeks, at which time it is removed simply by traction on the free end

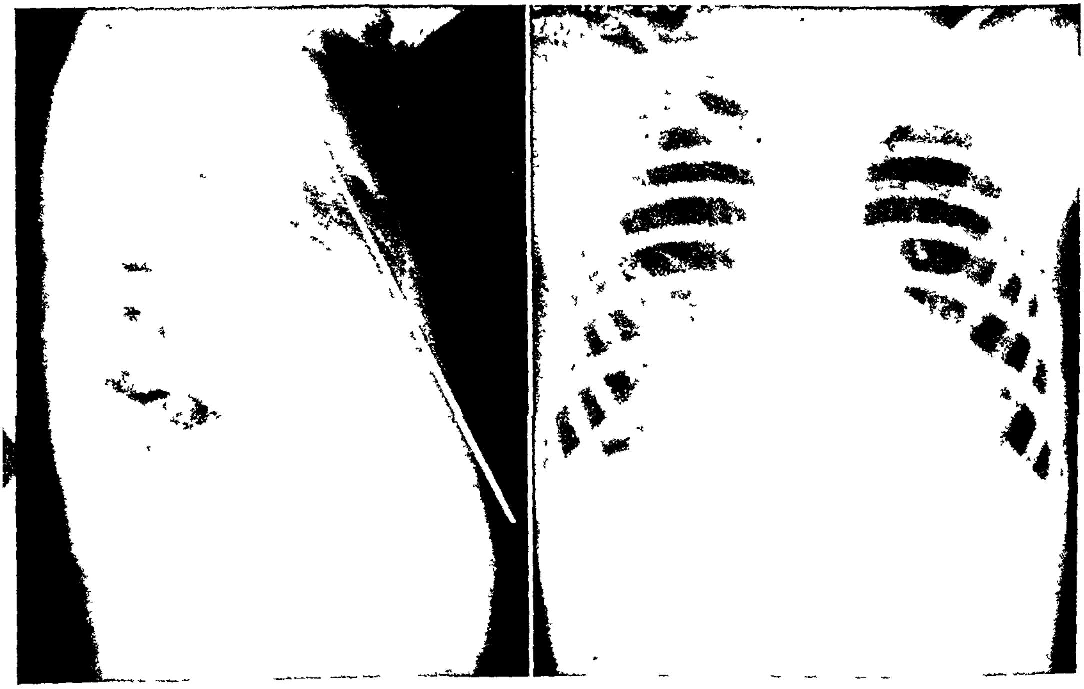
The elevation is sure and under accurate control until finally released It is painless to the patient and introduces no new liability to the desired end result At least no such factors have been encountered or visualized

## REFERENCES

1 Adams, H D Costosternoplasty with Rib Strut Support for Funnel Chest in Adults Lahey Clin Bull 7 111 116, 1951
2 Brodkin, H A Congenital Anterior Chest Wall Deformity of Diaphragmatic Origin, Dis Chest 24 259 277, 1953
3 Brown, A L Pectus Excavatum (Funnel Chest), J Thoracic Surg 9 164, 1939
4 Dailey, J E Repair of Funnel Chest Using Substernal Osteoperiosteal Rib Graft Strut, J A M A 150 1203 1204, 1950
5 Dorner, R A, Kell, P G, and Schissel, D J Pectus Excavatum J Thoracic Surg 20 444 453, 1950
6 Gross, R The Surgery of Infancy and Childhood, Philadelphia, 1953, W B Saunders Company, pp 753 761
7 Lester, C W Funnel Chest Its Cause, Effect, and Treatment, J Pediat 37 224 230, 1950
8 Meyer, Ludwig Quoted by Oschner and DeBakey [9]
9 Oschner, A, and DeBakey, M Chone Chondrosternum, J Thoracic Surg 8 469 508, 1938
10 Ravitch, M The Operative Treatment of Pectus Excavatum, Ann Surg 129 429 444, 1949
11 Sauerbruch, F Quoted by Oschner and DeBakey [9]
12 Sweet, R H Pectus Excavatus Ann Surg 119 922 1944
13 Woillez Quoted by Oschner and DeBakey [9]

maintained from 2 to 3 weeks. It may not be necessary after the first ten days but the additional period does no harm and since there is no discomfort, it is well tolerated and the additional interval probably allows further stabilization of the anterior chest wall and sternum. Up to ten days the external traction is considered essential because of the relative instability of the chest after resection of all the deformed cartilages. This, plus the "overcorrection" compensates for the paradoxical motion present in this area if traction is released. The 3/32 inch pin is removed quickly and simply by twisting the pin slightly, or if necessary, the hand drill can be attached and reverse-turned 1 or 2 cycles after which it will slip out readily.

This operative technique has now been used three times with good results in children, and the entire problem and misgivings of external traction has been simplified thereby. It was used also on one occasion in an adult, but was removed after four days because of a series of inexplicable complications unrelated to the pin, and probably triggered by the complications of an operative pneumothorax, and a postoperative empyema, so no conclusions can be drawn one way or another relative to its use in adults. It does seem, however, to be most ideally suited to children.



*A.* *B.*

Fig. 6.—Anteroposterior and lateral x-rays taken in the postoperative period showing pin in position. Wire traction applied to inferior end and suspended to wire ladder or other metal arch.

## SUMMARY

It is now generally recognized that pectus excavatum is a deformity of considerable physiologic significance, and that anatomic correction is desirable in nearly all cases.

Many techniques have been used in the evolution of a completely satisfactory surgical procedure, but a standardized procedure has been slow to appear due (1)

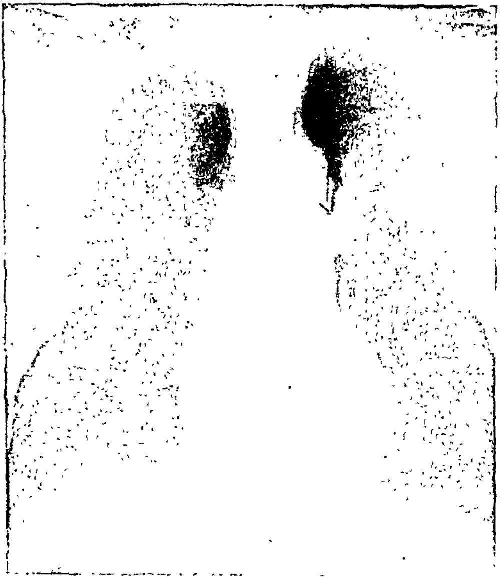


Fig. 1.—Planigram showing a frozen hilum.

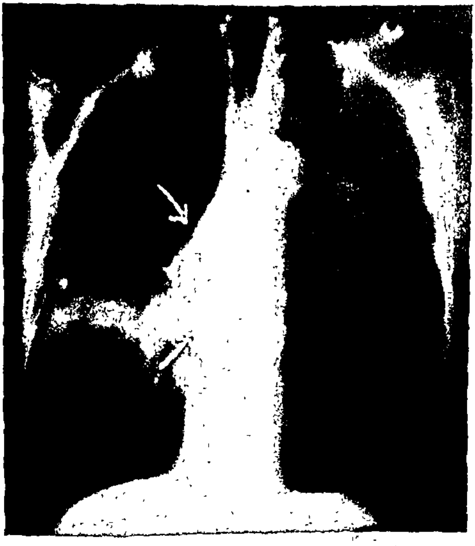


Fig. 2.—Planigram showing encroachment on carina [illegible] tumo

# CANCER OF THE LUNG

## AN ANALYSIS AND EVALUATION OF 100 CONSECUTIVE CASES

CLARENCE A. BISHOP, M.D.
LITTLE ROCK, ARK.

AN ANALYSIS of 100 consecutive cases of cancer of the lung is presented. This series includes only patients seen in the five-year period from December, 1950, to December, 1955. Though small in number, it is felt that the cases are quite representative. Every patient has had a complete follow-up.

In this series, 59 patients were considered to be not operable on admission to the hospital. The term "operable" as used herein is defined as meaning that the tumor (and nodes) may be extirpated en bloc dissection without cutting into or through grossly visible cancerous tissue.

Lesions were considered to be not operable when there was physical evidence of: (1) distant metastasis, (2) spread of the tumor to cervical lymphatics, as evidenced by a positive scalene node and fat pad biopsy, (3) encroachment by the tumor upon the carina and trachea, as evidenced by bronchoscopy and biopsy, (4) invasion of mediastinal structures, as evidenced by Horner's syndrome, tracheoesophageal or bronchoesophageal fistula, vocal cord paralysis, or phrenic nerve paralysis, and (5) pleural fluid positive for malignant cells.

Lesions were considered to be not operable when there was roentgenographic evidence of nonresectability by planigram and bronchogram, showing: (1) a "frozen" hilum as evidenced by a dense hilar mass obscuring the hilar components, (2) encroachment of the tumor and metastatic nodes upon the carina and trachea, (3) submucosal infiltration at the carina (by bronchogram), (4) paratracheal nodes extending up into the root of the neck, (5) invasion of mediastinal structures, and (6) evidence of metastasis to the opposite lung or nodes (Figs. 1 through 8).

For analytic as well as treatment purposes, the patients were divided into four groups:

*Group 1.*—Far-advanced hopeless cases in which there were bony or other distant metastasis. There were 41 patients in this group. None are now alive. Approximately one half of the patients in this group were given nitrogen mustard and/or deep x-ray therapy. The mean (not average) survival time of this group was two months following admission to the hospital for both the treated and untreated cases.

*Group 2.*—In this group, patients were considered to be nonresectable, but without immediate evidence of distant metastasis. There were 18 patients in

---

From the Surgical Service of Veterans Administration Hospital, Little Rock, Ark.
Received for publication Feb. 14, 1956.

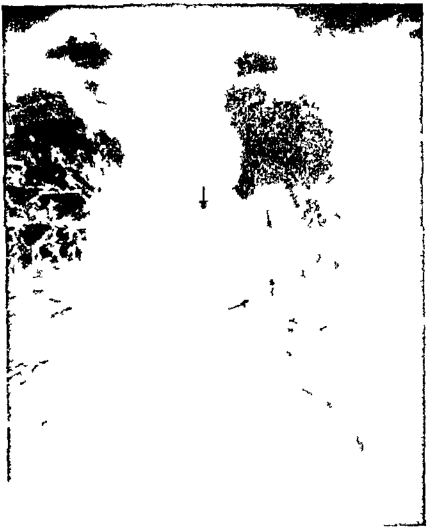


Fig 4.—Bronchogram of a recent patient deemed inoperable Planigrams and bronchogram reveal a frozen hilum and well demonstrated submucosal and paratracheal infiltration

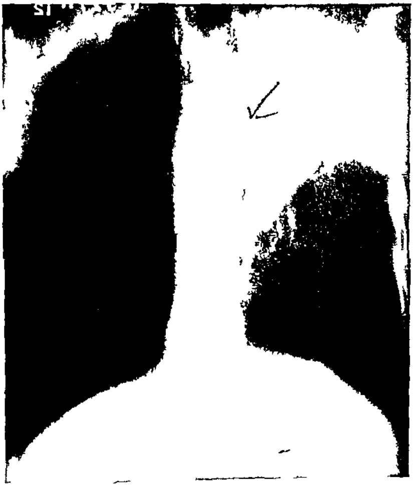


Fig 5A.—Planigram of patient with vocal cord paralysis showing invasion of mediastinal structures Survival time was 12 months

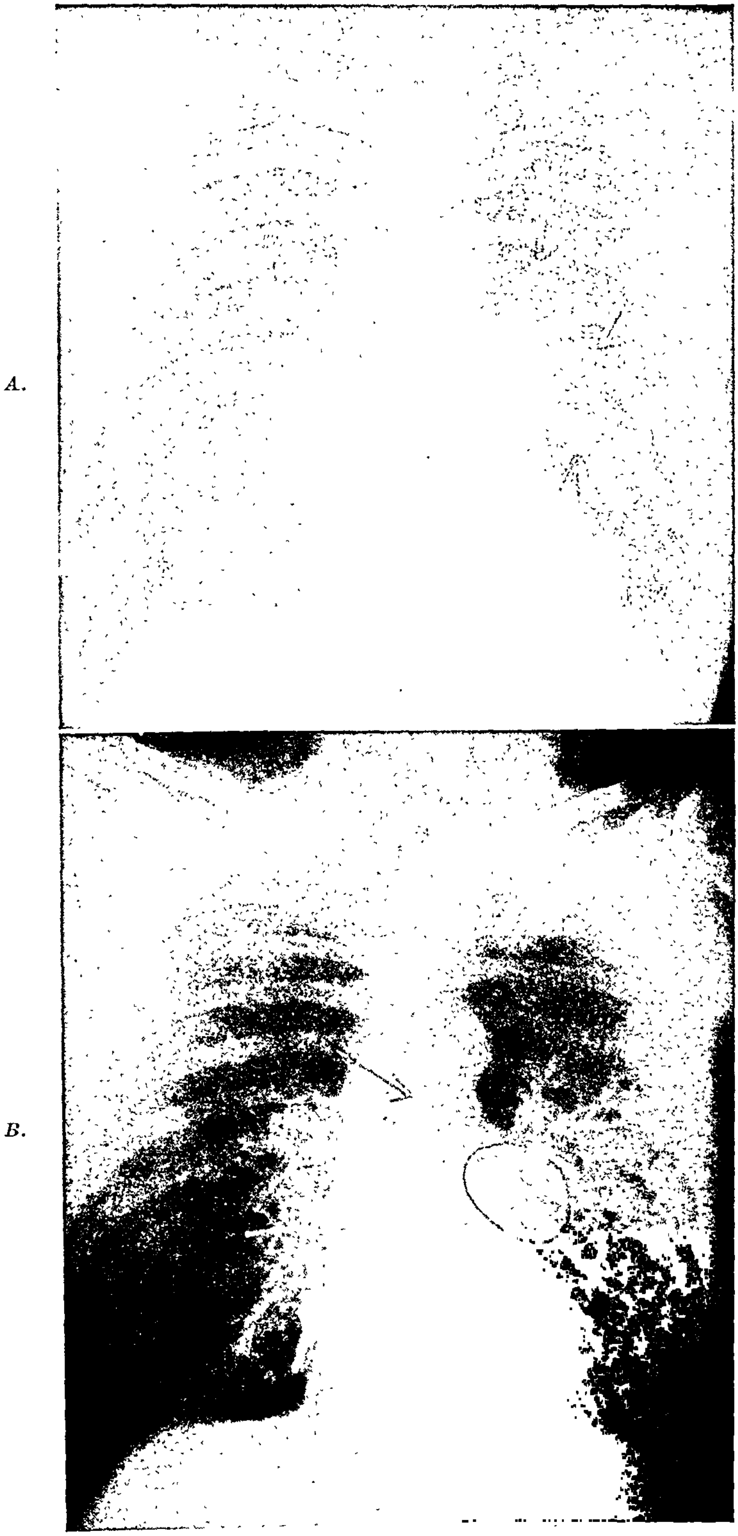


Fig. 3.—*A*. Posteroanterior view of patient with bilateral nodal metastasis. *B*, Bronchogram of same patient revealing submucosal infiltration at carina and into trachea. Lesion by planigrams and bronchogram was deemed inoperable. Survival time was 13 months following x-ray and nitrogen mustard therapy.

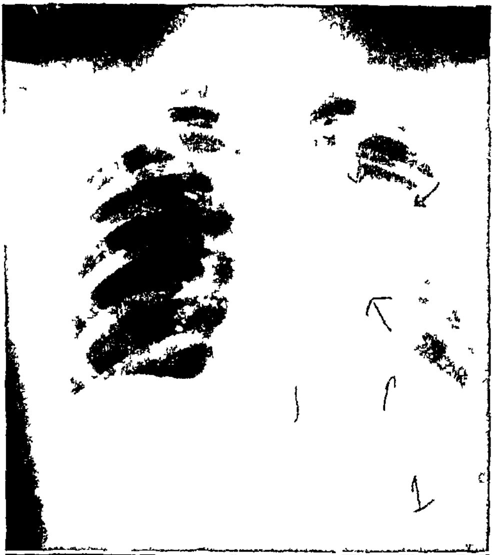


Fig 6

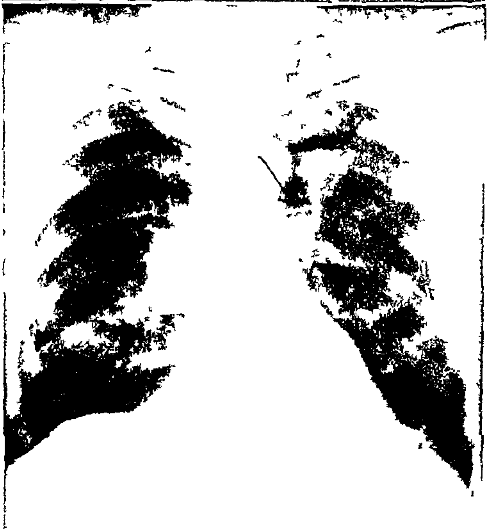


Fig 7

Figs 6 and 7—Posteroanterior views of 2 patients with bilateral nodal metastasis proved at post mortem examination

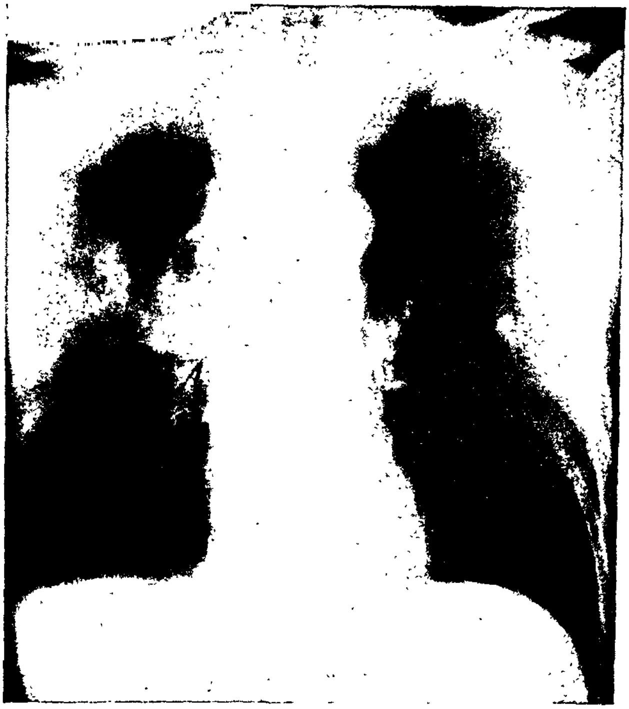


Fig. 5*B*.—Planigram of a patient with vocal cord paralysis showing invasion of mediastinal structures. Patient was treated with x-ray and nitrogen mustard. Survival time was 13 months.

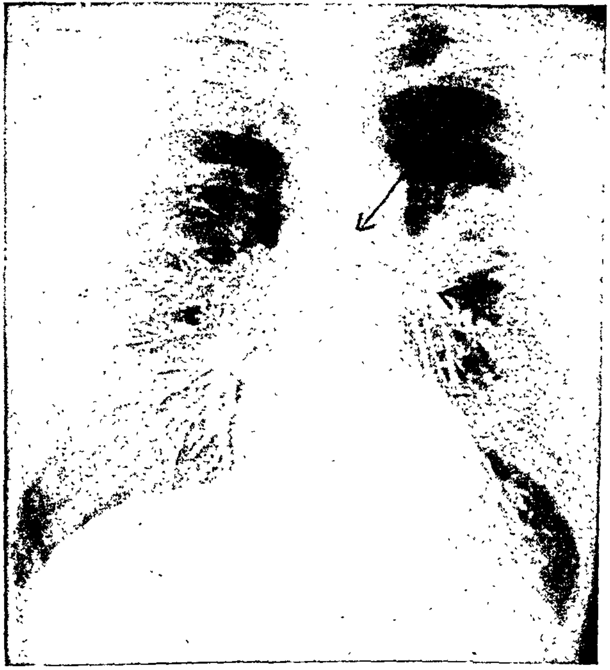


Fig. 5*C*.—Bronchogram (and planigrams) inconclusive of nonoperability. At surgery, this lesion was not resectable (frozen hilum). Post-mortem examination, in four months, revealed submucosal (and paratracheal) infiltration far up into trachea.

metastasis at present (An alveolar cell carcinoma for which a lobectomy was done because of a low respiratory reserve A positive biopsy was recently obtained at the carina ) (Tables I and II )

TABLE I GROUP 4 RESECTED CASES STILL ALIVE

| NO OF PATIENTS | TIME (YEARS) | PROCEDURE | PATHOLOGY |
|---|---|---|---|
| 1 | 5 | Lobectomy | Squamous cell carcinoma |
| 1 | 4¼ | Lobectomy | Malignant adenoma |
| 3 | 3 | Pneumonectomy | Oat cell carcinoma |
| 3 | 2½ | Pneumonectomy | Epidermoid carcinoma |
| 2 | 1½ | Pneumonectomy | Epidermoid carcinoma |
| 1 | 1¼ | Lobectomy | Alveolar cell carcinoma |
| 4 | 1 | Pneumonectomy | Epidermoid carcinoma |
| 1 | 1 | Lobectomy | Adenocarcinoma |
| Total 16 | | | |

TABLE II GROUP 4 RESECTED CASES NOT ALIVE

| NO OF PATIENTS | TIME (MONTHS) | PROCEDURE | PATHOLOGY | CAUSE OF DEATH |
|---|---|---|---|---|
| 1 | 22 | Pneumonectomy | Epidermoid carcinoma | Carcinomatosis |
| 1 | 12 | Pneumonectomy | Epidermoid carcinoma | Carcinomatosis |
| 1 | 12 | Pneumonectomy | Epidermoid carcinoma | Coronary disease (no evidence of carcinoma at post mortem examination) |
| 1 | 8 | Pneumonectomy | Epidermoid carcinoma | Carcinomatosis |
| 1 | 1 week | Pneumonectomy | Epidermoid carcinoma | Hospital death, atelectasis and pneumonia |
| Total 5 | | | | |

## DISCUSSION

From the mean survival time in months, it is immediately apparent that best results are obtained when the primary tumor is resected This has been proved over and over again No further comment will be made except to surmise that the five year survival rate in this series will not be much different from the many other reported series

It is equally apparent that by exploratory operations in nonresectable patients we are reducing the mean expectancy of life, in months, of these patients This is forcefully demonstrated by the less than four months mean survival time of our patients after fruitless thoracotomy, whereas, a presumably further advanced group with obvious contraindications to surgery survived a mean of ten months and in no instance survived less than five months when surgery was withheld Repeating the words of Brantigan, "there is a great obligation to withhold operation on patients with signs of nonresectability " We must use every available means to determine those lesions which are not resectable before exploration We cannot explore routinely every patient with cancer of the lung without a reasonable and thorough evaluation of resectability beforehand

There is no longer any doubt that there is a marked increase in the incidence of carcinoma of the lung It behooves us not only to increase the five year this group. Surgery was withheld and all of these patients were treated with nitrogen mustard and/or deep x-ray therapy. Seven patients are still alive, one for thirty months, 3 for eighteen months, one for twelve months, one for seven months, and one for five months. Of the 11 patients dead, the mean survival time was ten months, with a low of five months and a high of thirty-nine months.

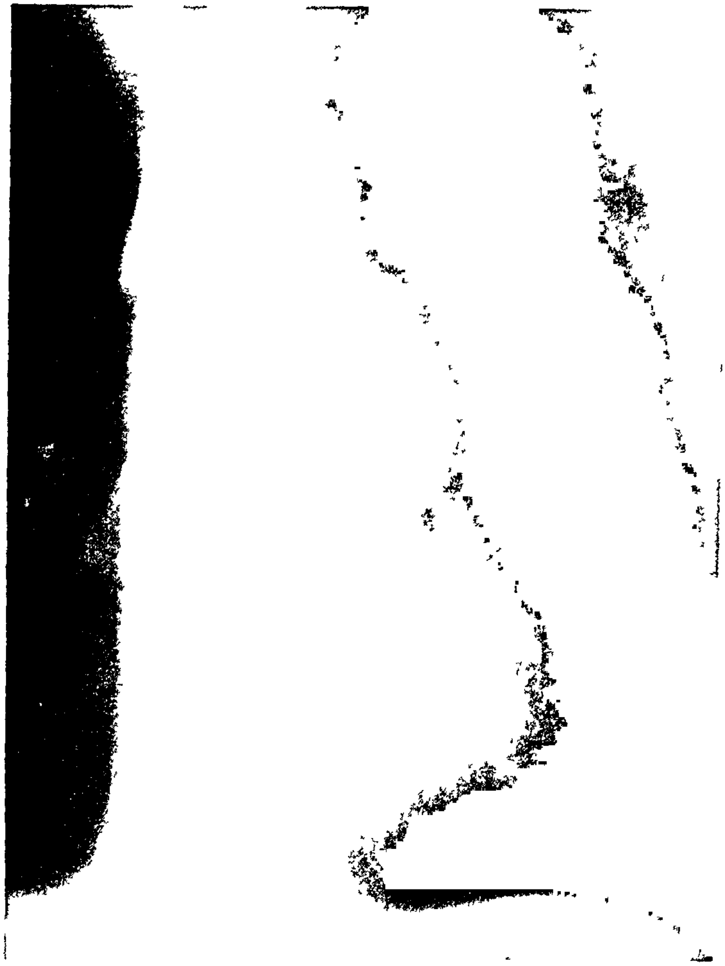


Fig. 8.—Planigram showing a frozen hilum and invasion of mediastinal structures. Note displacement of the left main bronchus by tumor. Survival time was 39 months after x-ray and nitrogen mustard therapy.

*Group 3.*—In this group, preoperatively, roentgenograms were not conclusive of inoperability. These patients all had exploratory operations and were found to be nonresectable. This group, numbering 20 patients, was treated postoperatively with nitrogen mustard and/or deep x-ray therapy. None of this group is now alive. The mean survival time in 16 cases was under four months. Two patients survived thirteen months, one patient twelve months, and one for nine months.

*Group 4.*—Twenty-one patients in whom resection was performed. Sixteen of the 21 are still alive. Only one of this group has a known recurrence or survival rate of operable patients, but also to increase the life expectancy and comfort of *all* patients with cancer of the lung, including the inoperable ones. This means a stricter evaluation of patients, withholding surgery on patients where lesions are by roentgenograms, etc., obviously not resectable.

There is growing evidence that dissection of, and biopsy of, nonresectable lesions leads to rapid and uncontrollable spread of cancer, not only in carcinoma of the lung, but in carcinoma of other organs as well, notably carcinoma of the head of the pancreas as one example. Everyone agrees that needle biopsy of inoperable tumors should not be done because of fear of spread of the tumor through the chest wall or adjacent tissue, with rapid generalized dissemination. With the chest open, one cannot biopsy a lesion without opening channels for spread or contaminating the chest cavity.

In this series, planigrams and bronchograms have been the most reliable sources of determining resectability, preoperatively. Planigrams in a semioblique position have, on occasion, been helpful in visualizing mediastinal invasion when normal vertical planigrams were inconclusive (Figs. 9 and 10). We have visualized bronchogenic carcinoma by bronchogram (submucosal infiltration) in a few cases not seen by the bronchoscopist, as well as in those cases impossible to be seen by bronchoscopy. We have been thoroughly disappointed with the use of routine scalene node and fat pad biopsy. A negative report has been received too often when the lesion was shown to be grossly not resectable by planigram, with metastasis to opposite lung, etc. We have never had a positive scalene node biopsy when roentgenograms were inconclusive of resectability. (Lesions that proved to be inoperable on exploration.) Angiocardiograms have not been used to determine resectability preoperatively. Mediastinal node biopsy has not been done in sufficient number to determine its value.

It is realized that many surgeons do not consider vocal cord and phrenic nerve paralysis, as well as invasion of the carina and trachea, evidence of nonresectability. In the past, we have found that when those conditions were present, although the primary tumor was removed in some cases without leaving visible cancer behind, there apparently was blood-borne dissemination already present in every instance, as evidenced by the early appearance of metastatic lesions postoperatively. Palliative resections for uncontrolled pain, abscess, invasion of chest wall, etc., are obvious necessities in some cases and have no place in this discussion.

Symptoms suggestive of cancer of the lung, as emphasized by Ochsner and others, are symptoms of complications. They are late symptoms and are probable evidence per se of a low five-year *expected* survival rate.

Surgery, to be effective, must be early. We must operate on suspicion of cancer. As emphasized by Rigler, Overholt, and others, it is possible to demonstrate a lesion in the lung by roentgenogram long before symptoms arise. Lack of a histologic diagnosis should not delay exploration when roentgenograms suggest cancer of the lung. Does one do harm by early exploration of (on suspicion of cancer) (1) hamartoma, (2) adenoma, (3) solitary [illegible]

Fig. 9.

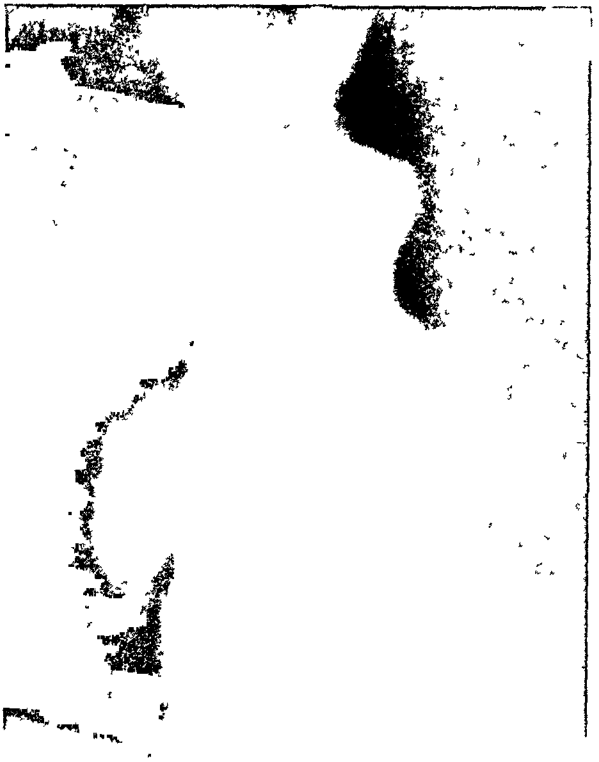


Fig. 10.

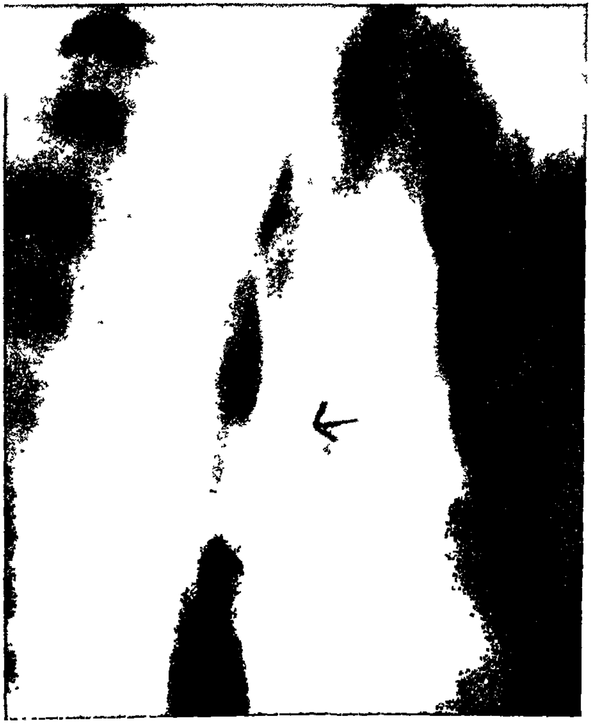


Fig. 9.—Planigram in routine position inconclusive of inoperability. Films taken elsewhere prior to admission were available and revealed no change in the nodes visible in the left hilum in five years.

Fig. 10.—Planigram of same patient in semi-oblique position reveals large sub-carinal and mediastinal nodes extending to opposite (left) side.

# THE IMMEDIATE POSTOPERATIVE COMPLICATIONS OF THORACIC SURGERY IN PULMONARY TUBERCULOSIS

DAVID E THOMAS, LIEUTENANT COLONEL, MC, USA
DENVER, COLO

## INTRODUCTION

WITH the advent of excisional surgery for pulmonary tuberculosis, the employment of such modes of therapy as extensive thoracoplasties, pneumoperitoneum, pneumothorax, or phrenic crush has waned almost to the point of disappearance

The proper selection of patients for surgery is in a state of flux at the present time Such factors as the type of disease which is best handled by drugs alone and the incidence of relapse following drug therapy compared to the morbidity and mortality of surgery must all be carefully weighed in making a decision for or against excisional surgery

There is a paucity of articles in the literature dealing with the postoperative complications of thoracic surgery in tuberculosis and no really exhaustive analysis of the problem

## REVIEW OF RECENT LITERATURE

Francis,[1] in an evaluation of the first 100 cases subjected to excisional surgery at Harefield Hospital in England, showed 90 per cent good results in the excision of large caseonodose residuals and only 64 per cent good results in the remainder, many of which represented problem cases with associated difficulties Thirteen fistulas occurred in the latter group and there were four operative deaths Spreads and reactivation of disease occurred in 6 patients soon after surgery Some adverse prognostic factors were considered to be inadequate treatment, unstable disease, inadequate localization of disease, endobronchitis, cavity opened at surgery, and positive sputum

Douglass and his colleagues[2] reviewed 589 consecutive pulmonary resections, of which 110 were classified as salvage cases while the remainder were elective Sixteen deaths occurred within sixty days after surgery, one third of the salvage cases and one fifth of the elective cases were complicated No attempt to correlate complications with significant factors was made

Decker and his associates[3] analyzed 355 resected and 109 drug treated patients and showed conclusively the value of surgery in the open positive and the open negative case An over all 0 6 per cent operative mortality and a complication rate of 12 per cent for the open positive case compared to 6 per cent and

---

From the Department of Surgery Fitzsimons Army Hospital Denver Colo
Received for publication Feb 17 1956

lesion, (4) lipoid pneumonia, or (5) an unexpansible atelectatic segment? (Other lesions besides carcinoma encountered on early surgery.) How does one rule out cancer clinically?

No patient over forty years of age should be discharged after a bout of pneumonitis without a follow-up roentgenogram of the chest. Age per se is no contraindication to surgery. Two of our patients still alive and well after resection are now over eighty years of age, having been seventy-nine years old at the time of resection.

## CONCLUSION

1. Every available means should be used preoperatively to determine resectability of lung cancers.
2. Inoperable cases without detectable distant metastasis may be palliated by use of nitrogen mustard and deep x-ray therapy if surgery is withheld.
3. In explored, but nonresected cases, there should be minimal disturbance of involved tissue to prevent dissemination of the tumor and rapid deterioration of the patient.
4. Surgery, to be effective, must be early. We must operate on suspicion of cancer—before symptoms arise—by detection of lesions too small to be diagnosed preoperatively, through routine *periodic* roentgen survey of the chest.

## REFERENCES

Ariel, I. M., Avery, E. E., Kanter, L., Head, J. R., and Landston, H. T.: Primary Carcinoma of the Lung, Cancer 3: 229-239, 1950.

Bloomer, W. E., and Lindskog, G. E.: Bronchogenic Carcinoma, Cancer 4: 1171-1175, 1951.

rantigan, O. C., and Hadidian, C. Y.: Cancer of the Lung, Maryland M. J. 4: 91-97, 1955.

rnett, W. E., Rosemond, G. P., Hall, J. H., and Caswell, H. T.: Carcinoma of the Lung, Arch. Surg. 61: 1052-1057, 1950.

hurchill, E. D., Sweet, R. H., Soutter, L., and Scannell, J. G.: The Surgical Management of Carcinoma of the Lung, J. THORACIC SURG. 20: 349-358, 1950.

DeBakey, M. E., Ochsner, A., and DeCamp, P. T.: Primary Carcinoma of the Lung, Surgery 32: 877-891, 1952.

Forster, E., Sichel, D., and Roegel, E.: Transversoaxial Tomography as a Valuable Help in Estimation of Operability of Pulmonary Cancer, J. THORACIC SURG. 27: 593-602, 1954.

Gibbon, J. H., Allbritten, F. F., Templeton, J. Y., and Nealon, T. F.: Cancer of the Lung—An Analysis of 532 Consecutive Cases, Ann. Surg. 138: 489-501, 1953.

Harken, D. E., Black, H., Clauss, R., and Farrand, R. E.: A Simple Cervicomediastinal Exploration for Tissue Diagnosis of Intrathoracic Disease, New England J. M. 251: 1041-1044, 1954.

Ochsner, A., DeCamp, P. T., and Ray, C. J.: Bronchogenic Carcinoma With Emphasis on Early Diagnosis, Geriatrics 9: 15-19, 1954.

Overholt, R. H., and Schmidt, I. C.: Silent Phase of Cancer of the Lung, J. A. M. A. 141: 817-821, 1949.

Overholt, R. H., and Schmidt, I. C.: Survival in Primary Carcinoma of the Lung, New England J. M. 240: 491-497, 1949.

Rigler, L. G.: Roentgen Examination of the Chest, J. A. M. A. 142: 773-777, 1950.

Rigler, L. G., O'Loughlin, B. J., and Tucker, R. C.: Significance of Unilateral Enlargement of the Hilus Shadow in the Early Diagnosis of Carcinoma of the Lung, Radiology 59: 683-693, 1952.

Slesser, B. V., Britt, R. G., and Freer, J. L.: Assessing Inoperability of Bronchial Carcinoma by Angiocardiography, Thorax 9: 91-99, 1954.

Watson, W. L.: Radical Surgery for Lung Cancer, Surg. Clin. North America 33: 401-414, 1953.

TABLE I. DISTRIBUTION OF CASES

| | AGE | 0-10 | 10-20 | 20-30 | 30-40 | 40-50 | 50+ | TOTAL |
|---|---|---|---|---|---|---|---|---|
| Negro | Male | 1 | | 6 | 1 | | | 8 |
| | Female | | 1 | 5 | 4 | | | 10 |
| White | Male | 1 | | 25 | 23 | 5 | | 54 |
| | Female | | 1 | 12 | 9 | 1 | 1 | 24 |
| Mongolian | Male | | | | | | | 0 |
| | Female | | | 3 | 1 | | | 4 |
| Subtotal | Male | 2 | | 31 | 24 | 5 | | 62 |
| | Female | | 2 | 20 | 14 | 1 | 1 | 38 |
| | Total | 2 | 2 | 51 | 38 | 6 | 1 | 100 |

The 100 patients involved in this report had a total of 146 anesthetics and 182 procedures. Obviously, multiple procedures at one thoracotomy are far from unusual

Since this detailed breakdown renders the material difficult to analyze accurately, the cases were reconsidered on the basis of the definitive or most important procedure performed. It is admitted that this approach permits the entrance of the human equation and may cause the reader to consider the factor of bias. In order to make the study comprehensible, cases involving more than one operative procedure at one thoracotomy are considered together Table II lists all the procedures performed and Table III tabulates the most important procedure in each case. One can see that multiple procedures must be considered together in order to have statistical significance. Also, in general, if multiple procedures are required, the process is more severe. Segmental plus wedge resections are considered separately for reasons which will become clear later.

TABLE II. PROCEDURES PERFORMED IN 100 CASES

| | |
|---|---|
| Segmental resection | 57 |
| Lobectomy | 27 |
| Thoracoplasty | 42 |
| Wedge resection | 26 |
| Decortication | 14 |
| Pneumonectomy | 1 |
| Monaldi operation | 1 |
| Biopsy lymph node and lung | 1 |
| Unroof sinus tract and bronchopleural fistula, old | 1 |
| Partial scapulectomy | 1 |
| Exploratory thoracotomy for bronchopleural fistula or postoperative hemorrhage | 2 |
| Closed intercostal tube drainage | 5 |
| Incision and drainage of incision | 2 |
| Tracheotomy | 1 |
| Secondary closure of wound | 1 |
| Total | 182 |

Having described the material and stated the objectives we must delve into semantics to decide what is a complication. Should one consider any deviation from an utterly smooth postoperative course such as the necessity to perform a thoracentesis for a relatively small amount of [illegible], would have the rather ridiculous case complication rate of [illegible] per cent. visualization, being so much more revealing in the chest, permits t surgeon the questionable luxury of worrying over situations the

5 per cent for the open-negative and closed-negative case, respectively, was shown. It is their conclusion that operative morbidity and mortality were not in any way prohibitive. It is interesting to note that they state that the incidence of significant operative complications increased in direct proportion to the magnitude of the resection.

Falk and Tucker[4] reported seven deaths among 310 patients in the immediate surgical period. A total failure and relapse rate of 7 per cent was recorded and was correlated with positive sputum, open cavities, and unstable roentgenographic lesions. Bronchial tuberculosis was related to bacteriologic relapse, as was localized empyema at the site of resection and reactivation of unresected disease. Failures occurred chiefly in the far-advanced group.

Murphy,[5] in an analysis of 133 patients undergoing segmental resection, concluded that patients did better if operated upon while on their first course of treatment; open cavities decreased the possibility of success, as did the presence of positive sputum.

Bell[6] experienced 20 per cent expansion problems in 308 segmental resections and wedge resections. He considered that most of them did not represent bronchopleural fistulas but rather fibrotic remaining lung or remaining lung which was already distended and emphysematous and not normally capable of expansion. His conclusions regarding treatment agree, in general, with mine. An interesting addendum to this article is the statement that there have been no postresection spaces in patients in the last 75 thoracotomies performed since adopting the technique of approximating raw surfaces. A discussion of this technique is to be the subject of a later article.[7]

## ANALYSIS OF 100 CONSECUTIVE CASES

A detailed analysis of 100 consecutive cases leaving the Tuberculosis Ward of the Thoracic Surgery Service at Fitzsimons Army Hospital was made in an attempt to determine significant factors related to complications. This service offers a unique opportunity to evaluate the problem under average conditions, since the surgical staff is a cosmopolitan group drawn from all parts of the country and trained at various centers. Each surgeon is responsible for the postoperative care of his patients, so a variety of procedures and approaches are employed.

The clinical material is primarily military personnel on active duty and their dependents, with a few veterans. A majority of the patients were on the Surgical Service for but a few weeks or months, but one patient was with us for six years. For age, sex, and race distribution, see Table I.

Patients are selected for surgery by a joint Medical-Surgical Board. The case is presented by the patient's ward officer, discussion follows, and a decision is reached. The criteria for surgery are not rigid, but, in general, those patients with large caseonodose residuals (2 cm. or over in greatest diameter), open or closed cavities, persistently positive sputum, severe tuberculous bronchiectasis, thick pleural peel, or history of reactivation, particularly after prolonged drug therapy, are selected for thoracotomy. Of the 100 patients, only 12 were in the so-called salvage category. The percentage of far-advanced cases is much smaller in Fitzsimons Army Hospital than in the average public-supported sanatorium.

It is the general feeling that complications, particularly expansion problems, are more common with segmental resections. Actually, this procedure was employed in 34 per cent of the cases, accounted for 34 per cent of the case complications and 44 per cent of the expansion problems—a statistic which is probably significant but not imposing. Segmental plus wedge resection accounted for 12 per cent of the 100 definitive procedures and 50 per cent of the 12 patients developed complications. Six of the 8 complications were expansion problems. Why this procedure should develop more complications is not readily determinable in view of the low incidence of complications for wedge resection alone. One might postulate that a wedge is often taken concomitantly with a segment in order to obtain disease which transects the segmental plane. Inadequate closure of this area of additional resection would increase the possibility of expansion problems. The 16 cases in which multiple procedures were performed at one thoracotomy resulted in 4 case complications, an incidence practically identical with that for the group as a whole and a finding not in agreement with a previously quoted article.[3]

Complications by age, race, and sex are listed in Table V. The 20- to 30-year bracket comprised 51 per cent of the cases and 39 per cent of the complications, while the 30- to 40-year group represented 38 per cent of the cases and 56.5 per cent of the complications. Males account for 62 per cent of the case population and 65 per cent of the case complications. With 78 per cent of the cases, the white race accounted for 91 per cent of the complications. This would suggest that increasing age increases the incidence of complications.

TABLE V. MAJOR COMPLICATIONS BY AGE, RACE, AND SEX

| | AGE | 0-10 | 10-20 | 20-30 | 30-40 | 40 50 | 50+ | TOTAL |
|---|---|---|---|---|---|---|---|---|
| Negro | Male | | | | 1 | | | 1 |
| | Female | | | 1 | | | | 1 |
| White | Male | | | 4 | 9 | 1 | | 14 |
| | Female | | | 4 | 3 | | | 7 |
| Subtotal | Male | | | 4 | 10 | 1 | | 15 |
| | Female | | | 5 | 3 | | | 8 |
| Total | | | | 9 | 13 | 1 | | 23 |

The factor of residual disease on the operated side was considered. It is common practice in this institution to make no attempt to extend the scope of the surgical procedure to include small fibrocaseous and caseonodose residuals felt to be of no surgical significance. Forty-four patients had residual disease remaining and 11 of these developed complications. This indicates that remaining disease on the operated side does not influence the complication rate. Bilateral disease may increase the complication rate slightly, since there were 10 complicated cases in 30 with evidence of bilateral disease. Positive sputum at the time of surgery was present in 3 patients and 2 of these developed complications. Such a small number of cases is of no statistical significance but
a trend in agreement with the findings of Murphy.[5]

TABLE III. DEFINITIVE PROCEDURE

| | | |
|---|---|---|
| Segmental resection | | 34 |
| Lobectomy | | 18 |
| Segmental and wedge resection | | 12 |
| Wedge resection | | 10 |
| Decortication | | 6 |
| Pneumonectomy | | 1 |
| Unroof sinus tract and bronchopleural fistula | | 1 |
| Modified thoracoplasty | | 1 |
| Mediastinal lymph node and lung biopsy | | 1 |
| Lobectomy and thoracoplasty | 3 | 16 |
| Lobectomy and segmental resection | 2 | |
| Lobectomy, wedge resection, and thoracoplasty | 2 | |
| Lobectomy, wedge resection, decortication, and thoracoplasty | 2 | |
| Segmental, wedge resection, and thoracoplasty | 1 | |
| Decortication and wedge resection | 1 | |
| Lobectomy, decortication, and thoracoplasty | 2 | |
| Lobectomy and wedge resection | 1 | |
| Lobectomy, segmental resection, and thoracoplasty | 1 | |
| Lobectomy and decortication | 1 | |
| Total | | 100 |

surgeon is not in a position to know exists. So, for the purpose of this study, a complication is defined as any postoperative situation which requires re-tubing of the chest or re-operation, is life endangering, may have serious sequelae, or results in death. Using these criteria, the case complication rate is 23 per cent. Many of these patients had multiple complications. Table IV lists the 28 complications encountered by procedure.

TABLE IV. MAJOR COMPLICATIONS BY PROCEDURE

| | | COMPLICATIONS | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| NO. OF ASES | PROCEDURE | EXPANSION PROBLEM | HEMOTHORAX | EMPYEMA AND BRONCHOPLEURAL FISTULA | CARDIAC ARREST | SUBSCAPULAR ABSCESS | WOUND INFECTION | ATELECTASIS | TOTAL |
| 8 | Segmental resection | 7 | 1 | | 1 | | | | 9 |
| 3 | Lobectomy | 1 | | | 1 | | 1 | | 3 |
| 6 | Segmental and wedge resection | 6 | | 1 | | 1 | | | 8 |
| 1 | Wedge resection | 1 | | | | | | | 1 |
| 1 | Pneumonectomy | | | 1 | | | | | 1 |
| 1 | Lobectomy with thoracoplasty | | 1 | | | | | 1 | 2 |
| 1 | Lobectomy, decortication, wedge, and thoracoplasty | | | | | | | 1 | 1 |
| 1 | Segmental, wedge, and thoracoplasty | 1 | | | | | | | 1 |
| 1 | Decortication and wedge | | 1 | 1 | | | | | 2 |
| 23 | Subtotal | 16 | 3 | 3 | 2 | 1 | 1 | 2 | 28 |

As is expected, the expansion problems, pneumothorax, hydropneumothorax, and hemopneumothorax, constitute the major portion of the complications. Minor degrees of atelectasis were frequent, but responded readily to treatment and did not meet the definition of a complication.

TABLE VIII TECHNICAL ERRORS AND ACCIDENTS RELATED TO COMPLICATIONS

| ERROR OR ACCIDENT | COMPLICATION |
|---|---|
| 1 Broken gravity drainage bottle | *Staphylococcus aureus* empyema |
| 2 Remaining bronchus compromised at segmental resection | Expansion problem |
| 3 Pneumoperitoneum in right upper quadrant | Air embolus, cardiac arrest, death |
| 4 Tube drainage of hemothorax for 21 days | *Staphylococcus aureus* empyema, bronchopleural fistula |
| 5 Long bronchial stump at pneumonectomy | Mixed empyema, bronchopleural fistula |
| 6 Operated upon with skin superficially infected and while on high steroid dosage | Wound infection, severe |
| 7 and 8 Tube disconnected from Stedman pump | Expansion problem |

the present time, patients with underwater seal bottles are not permitted indiscriminate ambulation and are trained to clamp or pinch off the tube immediately if anything should happen to the gravity drainage bottle Plastic, unbreakable bottles would be a help

The one death in this series occurred in a patient given pneumoperitoneum in the right upper quadrant at the close of an upper lobe segmental resection A peculiar churning noise in the chest was noted, followed by cardiac arrest The chest was promptly reopened and massage started, but a spontaneous sustained heartbeat failed to return in spite of repeated electric shocks and the usual ancillary measures At autopsy, the heart showed evidence of thermal injury resulting from the electric shocks There was no evidence of trauma to the liver, but it was felt that the patient had had an air embolus, probably from injection of air into the liver or some vein in the area Pneumoperitoneum as an aid in space obliteration in upper lobe resections was given up as being of questionable value When used after lower lobe surgery at the present time, it is given in the left lower quadrant of the abdomen while the patient is awake in order to avoid the liver and in order to appreciate untoward occurrences more rapidly

Prolonged tube drainage has been accepted as a cause for empyema and our experience would indicate this is valid As a rule it is our practice not to leave a tube in the chest for more than 14 days, and if there is still evidence of air leak at this time, operative measures for correction are taken Failure to follow this procedure probably resulted in empyema after 21 days of closed tube drainage in one case

A previous regime in 1949 failed to follow the basic surgical tenet of excising the bronchus near the carina during pneumonectomy The inevitable bronchopleural fistula and mixed empyema which occurred led to a series of risky operative procedures which eventually closed the fistula and terminated the empyema, but the patient is a borderline respiratory cripple and still has a positive sputum from lesions in the opposite lung He would never have been selected for excisional surgery according to our present criteria

A severe wound infection occurred in a patient operated upon while on high cortisone dosage with his skin incompletely cleared from a Merthiolate dermatitis The error, actually, was one of judgment, since the procedure was

Salvage surgery was performed on 12 patients in an attempt to remove life-endangering foci of disease such as tension cavities and restore the patient to a status consistent with extra-institutional life. Complications developed in 50 per cent of these patients, the increase in incidence being of a similar order to that reported by Douglass and his colleagues.[2]

The duration of therapy did not influence the complication rate. The range for all cases was 1 to 36 months with an average of 9 months, while the average for complicated cases was 10 months with a range of 6 to 36 months.

It was felt that the microscopic evaluation of the degree of activity of the lesion might significantly correlate with the complication rate, so 46 cases were analyzed with the results shown in Table VI. Surprisingly, the incidence of complications is essentially identical in the 3 categories.

TABLE VI. CORRELATION OF ACTIVITY OF LESION WITH COMPLICATIONS

| | ALL CASES | COMPLICATED CASES |
|---|---|---|
| Highly active | 8 | 2 |
| Moderately active | 18 | 5 |
| Slightly active and inactive | 20 | 4 |
| Total | 46 | 11 |

The possible correlation of the pathologic diagnosis with an increased complication rate for a specific type of tuberculous lesion was sought. The statistics are presented in Table VII. Where there are sufficient cases to make comparison valid, no significant percentage differential appears.

TABLE VII. CORRELATION OF COMPLICATIONS WITH PATHOLOGIC DIAGNOSES

| ALL CASES | | COMPLICATIONS | PERCENTAGE |
|---|---|---|---|
| Caseonodose | 87 | 22 | 25 |
| Caseocavitary | 33 | 7 | 21 |
| *Bronchiectasis* | 23 | 5 | 22 |
| Endobronchitis | 23 | 6 | 26 |
| Pulmonary emphysema | 4 | 2 | 50 |
| Tuberculous lymphadenitis | 4 | 1 | 25 |
| Chronic nonspecific bronchitis | 5 | 3 | 60 |
| *Total* | 179 | 46 | 26 |

It is commonly felt that tuberculous endobronchitis is associated with an increased incidence of bronchopleural fistula in the early postoperative period. This is undoubtedly so in the grossly demonstrable lesion. The diagnosis of endobronchial tuberculosis in our patients was made on the basis of microscopic examination, often on the finding of only one minimal submucosal lesion or disease in the vicinity of a bronchocavitary junction. It is apparent that endobronchial tuberculosis so diagnosed does not increase the postoperative hazard.

The complications related to operative and postoperative errors and accidents are listed in Table VIII. These are worthy of discussion, since they are so closely related to the human factor and subject to improvement.

It was formerly the practice to permit patients to ambulate carrying their gravity drainage bottles in slings. This was discontinued when one patient developed a *Staphylococcus aureus* empyema secondary to breaking a bottle. At

## SUMMARY

1. One hundred consecutive cases of pulmonary tuberculosis leaving the Thoracic Surgery Service at Fitzsimons Army Hospital were evaluated with respect to postoperative complications.

2. In 23 cases, there were 28 complications, the most frequent being expansion problems.

3. The percentage incidence of expansion problems was greatest in segmental resections combined with wedges and slightly more common than the group average in segmental resections alone.

4. Multiple procedures at one thoracotomy did not increase the incidence of complications.

5. Incidence of complications increases with age.

6. Residual disease on the operated side does not influence the complication rate, but cases of bilateral disease show a slight increase.

7. Complications are more frequent in so-called salvage cases.

8. Pathologic diagnosis or degree of activity of the lesion does not influence the complication rate; nor does the duration of antituberculous therapy.

9. The microscopic diagnosis of tuberculous endobronchitis in the absence of grossly visible endobronchial disease is not associated with an increase in complications.

10. The human factor contributed to complications in 8 cases. It is in this zone that improvement should be sought most diligently.

11. The complications increase morbidity but are usually amenable to treatment and an acceptable end result is to be expected.

## REFERENCES

1. Francis, R. S.: Partial Resection for Pulmonary Tuberculosis, 100 Cases Analyzed, Tubercle 35: 238-244, 1954.
2. Douglass, R., Bosworth, E. B., Judd, J. M., and Chang, K. H.: Resection Surgery in Tuberculosis, Complications and After-History, J. Thoracic Surg. 29: 136-150, 1955.
3. Decker, A. M., Jr., Raleigh, J. W., and Welles, E. S.: The Coordination of Surgery and Combined Chemotherapy in the Treatment of Pulmonary Tuberculosis, J. Thoracic Surg. 29: 151-160, 1955.
4. Falk, A., and Tucker, W. B.: Resection Failures in Pulmonary Tuberculosis: A Follow-up Study of 310 Patients, Tr. Nat. Tuberc. A., 1954, pp. 254-259.
5. Murphy, J. D.: A Study of the Factors Influencing the Success of Segmental Resection for Pulmonary Tuberculosis, Tr. Nat. Tuberc. A. 1954, pp. 264-269.
6. Bell, J. W.: Management of the Postresection Space in Tuberculosis. I. Following Segmental and Wedge Resection, J. Thoracic Surg. 29: 649-657, 1955.
7. Bell, J. W.: Personal Communication.

poor wound healing known to occur in the presence of high cortisone dosage added to the presence of organisms in the superficially infected skin and operative site was a combination practically assuring catastrophe. The lesson is obvious—do elective procedures when conditions are ideal.

Finding the chest tube disconnected from its source of negative pressure on two occasions emphasizes the importance of employing tight connections supplemented by adhesive strapping and of educating ward personnel and patients in the proper supervision and care of chest drainage equipment. In each of our cases, the condition was discovered by the surgeon and there was no way to determine how long it had existed. Fortunately, the resultant pneumothorax responded to the usual treatment and empyema did not occur.

## DISCUSSION

Excisional surgery for pulmonary tuberculosis is far from a benign procedure. The mortality rate, 1 per cent in this study, is not a deterrent, but the morbidity and complications are of sufficient magnitude that one must consider the individual case carefully before submitting the patient to thoracotomy. Considering the problem just from the standpoint of postoperative complications in a group composed mainly of young and middle-aged adults, one may reach certain conclusions.

A case complication rate of 23 per cent is not to be taken lightly. However, a majority of the complications responded to therapeutic efforts for their correction and, in all but one patient who should not have been operated upon initially, a satisfactory end result was obtained. Many paid for their complication with a reduced vital capacity secondary to fibrosis or thoracoplasty. The salvage cases required more extensive surgery, were fraught with complications, demanded the best of all concerned, but were most rewarding in that a successful end result represented a person who could not otherwise have been returned to society.

As is so often the case, the human factor accounted for or contributed significantly to one third of the complications, and it is only in this sphere, as far as can be determined by this study, that we should expect to see improvement in the near future. Certainly, nothing was discovered in the evaluation of other factors which would influence selection of patients, operative approach, or postoperative care, in the expectation of reducing complications.

Considering the matter in all its aspects, the complications of excisional surgery in tuberculosis are more a worry and concern to the doctor than a reason for withholding surgery from the patient with a lesion which should be resected. While the morbidity is increased and further operative procedures are often required, the end result in the treatment of complications is almost uniformly satisfactory. With the passage of time and further study and evaluation, the indications for surgery will become more clear-cut and definite. The complication rate may well have a definite influence in determining these indications.

Access to the interior of the right atrium was made possible by the atrial well technique of Gross and associates.[9] An opening was made into the left atrium by thrusting the point of the dissecting scissors through the fossa ovalis. Using the septal punch designed by Griffin and Essex,[13] the interatrial septum was then completely excised.

The technique for enlargement of the defect is based on the anatomic relation of *the ventral wall of the right superior pulmonary vein to the dorsal wall of the right atrium.* As illustrated diagrammatically in Fig. 1, these structures are adherent and merge with the posterior part of the interatrial septum. The right superior pulmonary vein lies immediately dorsal to a very thick muscular horizontal ridge of tissue on the dorsal wall of the right atrium. This prominent ridge lying between the caval orifices is termed the intervenous crest[14] (crista intervenens or tubercle of Lower),[15] and its inferior crescent shaped border, which deepens the fossa ovalis, is known as the superior limbus of the fossa ovalis. In the dog, the intervenous crest is a much more prominent structure than that which is seen in the human heart. Excision of the intervenous crest and the ventral wall of the right superior pulmonary vein results in the orifice of the pulmonary vein being located at the posterolateral margin of the right atrium, and the tributaries of this vein, draining the apical and cardiac lobes of the right lung, then empty into the right atrium *at its posterolateral margin.* The *limiting margin of the atrial septal defect* so created is composed of the cut edge of the interatrial septum superiorly, ventrally, and inferiorly, and the left atrial orifice of the right superior pulmonary vein, dorsally. This uninjured dorsal rim would not be likely to scar and hence such a defect would be less likely to undergo reduction in size than defects which were the result of excision of the atrial septum alone.

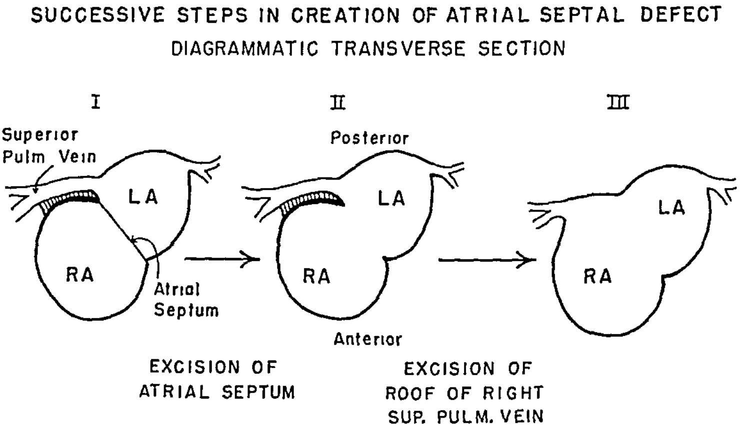


Fig. 1.—Diagrammatic transverse section through the atria at the level of the intervenous crest in the canine heart. The right and left atria are designated RA and LA respectively.

Panel I represents the undisturbed heart. The thickened posterior margin of the right atrium which is part of the ventral wall of the right superior pulmonary vein represents the intervenous crest which merges with the interatrial septum itself.

Panel II. The interatrial septum has been removed to produce an atrial septal defect.

Panel III. Excision of the intervenous crest and the ventral wall of the right superior pulmonary vein has resulted in a much larger defect. Note also that the uninjured dorsal wall of the right superior pulmonary vein is now the posterior margin of the defect.

*Hemodynamic Studies.*—A minimal period of one week was allowed for the animals to recover following production of the defect. Cardiac catheterization was carried out

# THE HEMODYNAMIC EFFECTS OF LARGE ATRIAL SEPTAL DEFECTS

## AN EXPERIMENTAL STUDY

DONALD E. BOWES, M.D.,* JOHN W. KIRKLIN, M.D.,** AND
H. J. C. SWAN, M.B., PH.D.***
ROCHESTER, MINN.

IN THE experimental surgical production of atrial septal defects the making of large defects and the spontaneous narrowing of small defects have been serious problems. As a result it has been difficult to study the effects of defects comparable in size to human atrial septal defects over any significant period of time. Spontaneous closure and marked narrowing of small defects were reported by Martin and Essex,[1] Little and associates,[2] Dodrill,[3] Leeds,[4] and Swan and associates.[5]

Permanent atrial septal defects were produced by Hanlon and Blalock,[6] the diameter of the resulting interatrial communications averaging 1.0 to 1.5 cm., and were localized to the superior and posterior part of the septum. Interatrial communications were also created by Kiriluk and associates,[7] and Moore and Shumacker.[8] The defects produced by the latter group had a mean diameter of 1.7 cm. Both groups of investigators stressed the importance of complete excision of the interatrial septum in order to achieve permanent defects. Using the atrial well technique of Gross and associates,[9] permanent atrial septal defects have been achieved by Gross and associates,[9, 10] Donald and associates,[11] and Silver and associates.[12] The majority of these studies were concerned with the techniques for the production and closure of atrial septal defects. Little and associates[2] studied the difference in atrial pressures. While certain of these investigators described hemodynamics, no data are available to determine the changes produced over a period of time by persistent interatrial communications.

This study reports a modification of previous techniques whereby large permanent atrial septal defects were produced in dogs and describes the hemodynamic effects of such lesions at intervals of one week to one year after operations.

### METHODS

*Surgical.*—Mongrel dogs weighing 17 to 32 Kg. were anesthetized with sodium pentobarbital (25 mg./Kg.), and thoracotomy was performed through the fourth or fifth interspace.

---

Abridgment of thesis submitted by Dr. Bowes to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Surgery.

The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

Received for publication Feb. 24, 1956.

*Fellow in Surgery, Mayo Foundation, Rochester, Minn.

**Section of Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minn.

***Section of Physiology, Mayo Clinic and Mayo Foundation, Rochester, Minn.

Indicator dilution curves[22] were recorded by a cuvette oximeter through which the femoral artery blood was permitted to flow following injection of T-1824 (Evans blue dye) into the chambers of the heart and great vessels. Such curves, which will be reported in a separate study, indicated the presence of a right-to-left shunt in many instances.

## RESULTS

*Technical Success.*—The procedure was successfully completed in 12 of 16 animals that underwent operation. One animal died on the fifth postoperative day from acute left heart failure due to mitral regurgitation, the anterior leaflet of the mitral valve having been damaged at operation. The remaining 11 animals had an uneventful postoperative period. None appeared incapacitated in any way. When the animals were anesthetized prior to cardiac catheterization, a moderately loud systolic murmur was heard in the pulmonic area in about one-half of them. This was probably associated with the tachycardia and quiet respiration accompanying pentobarbital anesthesia. One animal (No. 12) developed ventricular fibrillation at the start of the cardiac catheterization procedure and died before data were obtained. Of the 11 animals examined at necropsy to date all but one (No. 7) had large septal defects and anatomic connection of the right superior pulmonary vein to the right atrium. A typical example of the anatomic findings is illustrated in Fig. 2. Estimates at operation indicated that comparable defects were made in all animals except one (No. 7). This animal had a small atrial septal defect which measured 0.8 by 1.0 cm. This was due to incomplete excision of the right superior pulmonary vein area because of operative hemorrhage. Small defects are likely to occur when the septum is not completely excised and are most likely to be reduced further in size by hypertrophic scarring.

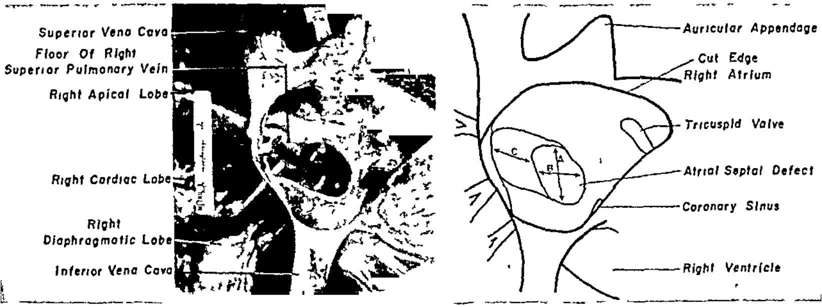


Fig. 2—Relation of veins from the right lung and other structures to a surgically created atrial septal defect in a dog (No 5) killed one year after production of the defect Probes placed in the veins draining the apical and cardiac lobes of the right lung enter the lateral portion of the right atrium A probe in the diaphragmatic lobe of the right lung can be seen in the left atrium through the atrial septal defect The prominent intervenous crest (crista divisens) has been completely removed. In the right panel the diameters of the defect are indicated by the arrows *A* and *B* (see Table I) while the length of the excised ventral wall of the pulmonary vein is indicated by the arrow *C*

with each animal under pentobarbital anesthesia. An endotracheal tube with an inflatable cuff was passed into the trachea. Intracardiac and arterial pressures were recorded by means of strain gauge manometers.[16] The zero reference for pressure measurement was a point halfway between the sternum and the table top with the animal supine. The oxygen saturation of the blood samples drawn from the chambers of the heart and great vessels was determined by photometric[17] and manometric methods.[18]

In each instance the sequence of the study was as follows: With the animal breathing air, the catheter was introduced via the jugular vein into the inferior vena cava, and blood samples were drawn through an oximeter from this position and, immediately following, from the right atrium and superior vena cava. Pressures were then measured in the right ventricle, pulmonary artery, pulmonary artery wedge, pulmonary vein, and pulmonary vein wedge positions. The endotracheal tube was then connected to a recording spirometer containing 100 per cent oxygen and providing for absorption of carbon dioxide on the expiratory airway. The oxygen consumption of the animal was recorded by means of this apparatus and, during the period of this measurement, blood samples were drawn from the femoral and pulmonary arteries. The catheter was then withdrawn from the pulmonary artery and advanced to the inferior vena cava. Blood samples were drawn through the cuvette oximeter from this location, following which the catheter was removed to the superior vena cava and a second sample was drawn from this site. The oxygen content of mixed venous blood ($C_{M.V.B.O_2}$)* can be expressed as

$$C_{M.V.B.O_2} = \frac{2S(ivc) + S(svc)}{3} (C_{Hb}),$$

where S(ivc) and S(svc) refer to oxygen saturation of inferior and superior caval blood, respectively, and $C_{Hb}$ is the oxygen capacity in cubic centimeters per liter of blood. The oxygen content and capacity ($C_{Hb}$) of the blood samples drawn from the femoral and from the pulmonary artery were determined by the method of Van Slyke and Neill[18] with the modification for oxygen capacity of Sendroy.[20] Pulmonary ($Q_p$) and systemic ($Q_s$) flows were determined according to the equations:

$$Q_p = \frac{V_{O_2}}{C_{P\,V\,O_2} - C_{P\,A\,O_2}},$$

$$Q_s = \frac{V_{O_2}}{C_{S\,A\,O_2} - C_{M.V.B\,O_2}}.$$

In these equations $C_{P.V.O_2}$ = oxygen content of pulmonary vein blood (c.c./L.).
$C_{P\,A.O_2}$ = oxygen content of pulmonary artery blood (c.c./L.).
$C_{S.A.O_2}$ = oxygen content of femoral artery blood (c.c./L).
$C_{M.V.B.O_2}$ = oxygen content of mixed venous blood (c.c./L.).

Assuming that the data of Wood and Cronin,[21] concerning the quantity of oxygen carried in physical solution when human subjects breathed 100 per cent oxygen, are applicable to the dog, the oxygen content of blood in the pulmonary vein was determined by adding 1.8 volumes per 100 c.c. of blood to the oxygen capacity of femoral artery blood. A right-to-left shunt was considered present if the difference between oxygen content and capacity of femoral artery blood was less than 1.6 volume per 100 c.c. of blood.

On a number of occasions the determination of values for oxygen content and oxygen capacity was unsatisfactory either because the oxygen capacity of samples of blood drawn simultaneously from the pulmonary artery and the femoral artery differed in a random fashion by a value greater than 0.4 volumes of oxygen per 100 c.c. of blood or because the oxygen content of femoral artery blood exceeded the oxygen capacity of femoral artery blood by more than 2.2 volumes of oxygen per 100 c.c. of blood. Under such circumstances the values were discarded and the studies were repeated.

---

*The symbols used are adapted from those recommended by Pappenheimer and associates.[19]

months following operation were found to have an elevation of pressure above the value observed at three weeks and above the range obtained in the normal dogs.

Table II. Intracardiac and Intravascular Pressures (mm. Hg) Recorded in 10 Dogs Studied 1 to 6 Weeks Following Creation of Large Atrial Septal Defects

| ANIMAL | WEEKS POST-OPERATIVE | RIGHT ATRIUM | RIGHT VENTRICLE | PULMONARY ARTERY MAIN | PULMONARY ARTERY WEDGE | FEMORAL |
|---|---|---|---|---|---|---|
| 1 | 2 | 6/2 | 28/2 | 27/13 | 9/6 | 190/110 |
| 2 | 2 | 5/3 | 32/-1 | 28/12 | 12/8 | 195/130 |
| 3 | 1 | 10/2 | 33/0 | 24/13 | 13/9 | 200/135 |
| 4 | 2 | 5/-1 | 53/1 | 40/15 | 6/3 | 230/175 |
| 5 | 2 | 7/1 | 47/6 | 38/15 | 18/13 | 190/150 |
| 6 | 2 | 11/3 | 49/3 | 38/15 | 13/8 | 180/130 |
| 7 | 6 | 7/1 | 36/0 | 32/14 | 11/7 | 190/135 |
| 8 | 2 | 6/-1 | 27/-1 | 24/5 | 10/4 | 190/135 |
| 9 | 2 | 5/3 | 40/2 | 37/9 | 12/10 | 170/125 |
| 10 | 3 | 9/2 | 47/6 | 41/20 | 26/22 | 185/130 |
| Average | 2.4 | 7/1 | 39/2 | 33/13 | 13/9 | 190/135 |
| 13 Normal dogs | Average | 3/-1 | 27/-1 | 26/10 | 7/4 | 180/125 |
|  | Range | 0/-6 to 6/-1 | 23/-3 to 34/2 | 20/6 to 35/20 | 2/-4 to 11/8 | 160/92 to 235/175 |

It will be noted that the average systolic pressure in the main pulmonary artery was less than the average systolic pressure in the right ventricle in all the animals. This gradient of pressure, which averaged 6 mm. Hg and ranged from 1 to 13 mm. Hg, was found between the pulmonary artery and right ventricle in the experimental group, whereas on the average, no significant gradient was found in the normal group. One of the normal dogs had a gradient of 6 mm. Hg.

In Fig. 3, a photographic record obtained during withdrawal of the cardiac catheter from the main pulmonary artery to the right ventricle, the abrupt increase in pressure, which amounted to 11 mm. Hg, is clearly demonstrated as the catheter passed across the pulmonary valve. In other instances the systolic pressure in the right ventricular outflow tract equaled the systolic pressure in the main pulmonary artery, but an increase in pressure appeared to occur when the catheter was withdrawn to the mid-right ventricle or when the tip lay just within the tricuspid valve. The highest right ventricular pressures were usually recorded in this area. The right ventricular diastolic pressure was normal.

The pulmonary artery wedge pressure was slightly greater than the right atrial pressure. The average value (25/16 mm. Hg) for pulmonary vein wedge pressure (obtained from the right apical and cardiac lobe veins because of their accessibility to a cardiac catheter) was significantly less than the pulmonary artery pressure (33/13 mm. Hg).

In the 5 animals in which comparative data were obtained at three months (Table III), an increase in the average pressure values was found. This increase was principally due to the change in 3 animals in which the values obtained on the first study were within normal limits. The absolute values obtained for this group at three months did not differ significantly

TABLE I. VITAL DATA CONCERNING 16 DOGS WITH LARGE ATRIAL SEPTAL DEFECTS

| ANIMAL | WEIGHT (KG.) | KILLED OR DIED, WEEKS POSTOPERATIVE | DIMENSIONS OF ATRIAL SEPTAL DEFECTS, CM.* | | |
|---|---|---|---|---|---|
| | | | A | B | C |
| | | *Animals Catheterized* | | | |
| 1 | 17.8 | 52 | 1.7 | 1.3 | 1.5 |
| 2 | 24.5 | 52 | 2.0 | 1.8 | 1.5 |
| 3 | 19.0 | 6 | 1.6 | 1.6 | 1.5 |
| 4 | 25.0 | 12 | 2.2 | 2.2 | 1.2 |
| 5 | 19.7 | 51 | 1.8 | 1.8 | 1.8 |
| 6 | 16.0 | 19 | 1.5 | 1.7 | 1.5 |
| 7 | 17.0 | 6 | 0.8 | 1.0 | -- |
| 8 | 24.5 | 44 | 1.0 | 1.4 | 1.1 |
| 9 | 32.0 | 44 | 2.6 | 2.0 | 1.0 |
| 10 | 21.7 | Still alive | | | |
| | | *Animals Surviving Operation But Not Studied* | | | |
| 11 | 20.0 | 1 | 3.0 | 2.0 | 1.0 |
| 12 | 19.0 | 3 | 2.0 | 1.7 | 1.5 |
| | | *Animals Failing to Survive Operation* | | | |
| 13 | 18.5 | During operation | 3.0 | 3.0 | 1.5 |
| 14 | 20.0 | Less than 24 hrs. after operation | 3.0 | 3.0 | 1.5 |
| 15 | 19.8 | | 2.5 | 2.5 | 1.5 |
| 16 | 18.5 | | 2.5 | 2.0 | 1.0 |

*See Fig. 2 for representation of A, B, and C. A is the vertical (cephalocaudal) and B is the horizontal (dorsoventral) dimension. C is a measurement of the longitudinal axis of the exposed floor of the right superior pulmonary vein and represents the lateral distance from the location of the atrial septal defect to the point at which the vein enters the right atrium.

Measurements of the atrial septal defect proper and the floor of the right superior pulmonary vein are given in Table I. The difference between the animals that failed to survive the operation and the remainder suggested the possibility that the defects decreased in size in the first four weeks following operation.

*Hemodynamic Effects.—*

*Demonstration of arterialization in right atrium:* By means of a cuvette oximeter,[17] an instrument of high relative accuracy, the oxygen saturation of samples of blood drawn in rapid succession from the inferior vena cava, right atrium, and superior vena cava could be determined. With this sampling technique it was demonstrated that the oxygen saturation of blood in the right atrium exceeded that of mixed venous blood in 13 of 14 animals studied. The average values for oxygen saturation of blood from the inferior vena cava, superior vena cava, and right atrium were 74, 84, and 89 per cent, respectively, while the animals breathed room air, and 92, 96, and 96 per cent, respectively, while the animals spontaneously breathed pure oxygen.

*Pressures:* The values for intracardiac and intravascular pressures in each animal are given in Table II. For comparison, the average values for a group of normal animals studied by the same techniques and under similar conditions were assembled. The majority of the animals in the atrial septal defect group had elevated pressures at the first postoperative catheterization. Six of the animals were found to have right ventricular systolic pressures greater than the range found in normal dogs. In 4 of the animals the pressure was within the range of normal. However, 3 of these animals at three

TABLE IV. PULMONARY AND SYSTEMIC FLOW VALUES AND MAGNITUDE OF INTRACARDIAC SHUNTS IN 9 ANIMALS BREATHING 100 PER CENT OXYGEN STUDIED 1 TO 3 WEEKS FOLLOWING PRODUCTION OF THE ATRIAL SEPTAL DEFECT*

| ANIMAL | LITERS PER MINUTE | | SHUNTS, % | |
|---|---|---|---|---|
| | PULMONARY FLOW | SYSTEMIC FLOW | L → R | R → L |
| 1 | 6.5 | 4.1 | 54 | 27 |
| 2 | 6.2 | 6.9 | 39 | 45 |
| 3 | 4.2 | 5.2 | 12 | 29 |
| 4 | 16.3 | 4.2 | 74 | 0 |
| 5 | 7.0 | 4.5 | 44 | 13 |
| 6 | 6.8 | 3.0 | 56 | 0 |
| 8 | 4.4 | 4.4 | 0 | 0 |
| 9 | 3.6 | 3.9 | 3 | 10 |
| 10 | 7.2 | 6.3 | 40 | 32 |
| Average | 6.9 | 4.7 | 36 | 17 |

*Average value for cardiac output in 13 normal dogs of comparable weight (20.0 Kg.), breathing 100 per cent oxygen, was 3.2 L. per minute (range 1.8 to 4.4 L/min.)

monary flow was considerably elevated and the systemic flow was moderately elevated above the average for a group of normal dogs. The values showed a wide variability. In most instances the pulmonary flow was much greater than the systemic flow, indicating the presence of a left-to-right shunt, but right-to-left shunts were also calculated in many of the animals. The average left-to-right shunt was 36 per cent of pulmonary flow, and the average right-to-left shunt was 17 per cent of systemic flow.

While the animals were breathing 100 per cent oxygen from the recording spirometer, it was noted that large fluctuations in intracardiac pressure occurred, indicating a considerable variation in intrapleural pressure with respiration. Since this circumstance might well alter the magnitude and direction of intracardiac shunts, further calculations were undertaken. Utilizing the value for oxygen consumption obtained while the animals breathed 100 per cent oxygen and the values for oxygen saturation of the blood from the pulmonary and femoral arteries and the mixed venous blood while the animals were breathing air, estimates of pulmonary and systemic flows were made. Under this circumstance the pulmonary flow averaged 7.6 L. per minute and the systemic flow 3.0 L. per minute, and the magnitude of left-to-right and right-to-left shunts was 51 and 6 per cent, respectively. It would seem that these values might better represent the more usual dynamic situation

TABLE V. AVERAGE CHANGE IN FLOW VALUES AND INTRACARDIAC SHUNTS FROM 10 STUDIES AT 3 AND 10-12 MONTHS FOLLOWING CREATION OF ATRIAL SEPTAL DEFECT. EXPRESSED IN ABSOLUTE VALUES AND PERCENTAGE OF CHANGE

| NUMBER OF ANIMALS | BREATHING OXYGEN | | | | | | | | BREATHING AIR | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | PULMONARY FLOW | | SYSTEMIC FLOW | | SHUNT, % | | | | SHUNT, % | | | |
| | | | | | L → R | | R → L | | L → R | | R → L | |
| | MM. Hg | % | MM. Hg | % | MM. Hg | % | MM. Hg | % | MM. Hg | % | MM. Hg | % |
| | | | | | *At 3 Months* | | | | | | | |
| 5* | +0.3 | 4 | -0.1 | 2 | +4 | 7 | -3 | 17 | -10 | 21 | +8 | 100 |
| | | | | | *At 10-12 Months* | | | | | | | |
| 5† | +2.1 | 38 | +1.3 | 31 | +8 | 28 | -10 | 52 | -10 | 22 | +1 | 12 |

*Animals 1, 2, 4, 5, and 6.
†Animals 1, 2, 5, 8, and 9.

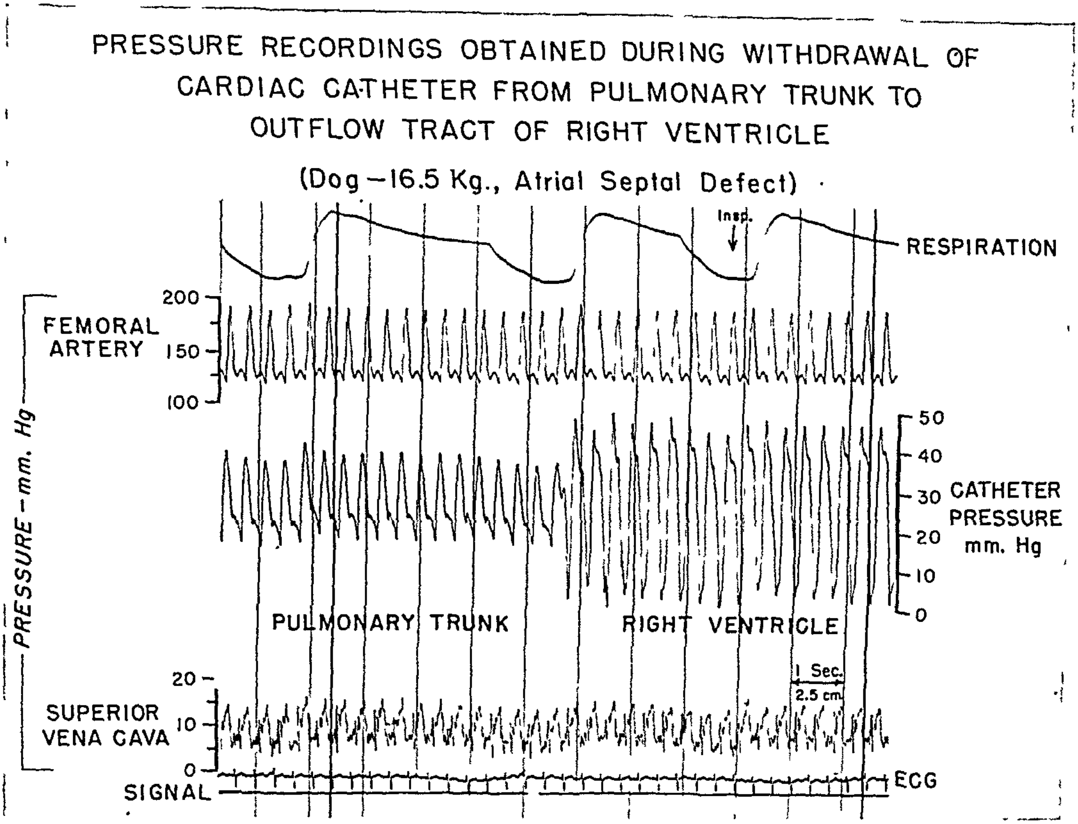


*Fig. 3.*—Photographic record of simultaneous pressures in the femoral artery, pulmonary artery, and superior vena cava. A cardiac catheter located just above the pulmonary valve was withdrawn under fluoroscopic control at the signal on the base line until it lay just below the pulmonary valve. Note the abrupt change in the systolic pressure as the catheter entered the right ventricle, in the absence of any change in heart rhythm or femoral artery blood pressure.

from the values obtained at the first study in the majority of the animals. There was no further change in the average values of the pressure in the animals studied ten to twelve months postoperatively (Table III).

TABLE III. CHANGE IN AVERAGE INTRACARDIAC AND INTRAVASCULAR SYSTOLIC PRESSURES IN ANIMALS STUDIED 3 MONTHS AND 10-12 MONTHS POSTOPERATIVELY

| NUMBER OF ANIMALS | RIGHT ATRIUM | | RIGHT VENTRICLE | | PULMONARY ARTERY | | | | FEMORAL | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | MAIN | | WEDGE | | | |
| | MM. Hg | % | MM. Hg | % | MM. Hg | % | MM. Hg | % | MM. Hg | % |
| | | | | | *At 3 Months* | | | | | |
| 5* | +3 | 43 | + 7 | 17 | +8 | 23 | 0 | 0 | +5 | 2 |
| | | | | | *At 10-12 Months* | | | | | |
| 5† | +1 | 14 | +10 | 24 | +8 | 23 | -1 | -9 | +7 | 3 |

*Animals 1, 2, 4, 5, and 6.
†Animals 1, 2, 5, 8, and 9.

*Pulmonary flow, systemic flow, and intracardiac shunts:* The values for pulmonary and systemic flow, and magnitude of intracardiac shunts for the group of 9 animals studied while breathing 100 per cent oxygen one to three weeks following operation are given in Table IV. The average value of pulto those just described. This gradient has also been found in cases of atrial septal defect in the human being[26] and is thought to be related to an increase in the volume of pulmonary flow.

The pulmonary artery wedge pressure exceeded the right atrial pressure in all instances. Although in certain comparisons there was a close agreement in the values obtained, there were large discrepancies in others. These findings are in agreement with those of Haddy and associates[27] in the dog and differ from those of Connolly, Kirklin, and Wood,[28] who demonstrated substantial agreement between pulmonary artery wedge and right atrial pressures in human patients. Furthermore, in the present study, agreement was not found between the pulmonary vein wedge pressure and the pulmonary artery pressure.

## SUMMARY

1. Large permanent atrial septal defects were produced in dogs by excision of the ventral wall of the right superior pulmonary vein and the adherent dorsal wall of the right atrium in addition to complete removal of the interatrial septum.

2. Intravascular and intracardiac pressures were elevated soon after production of the defect and showed no further change beyond three months postoperatively.

3. A gradient of pressure was found between the pulmonary artery and the right ventricle.

4. High pulmonary flow values, moderately large left-to-right shunts and small right-to-left shunts were demonstrated. No progressive increase in magnitude of left-to-right shunt or in the pulmonary artery pressure occurred in the months following creation of the defect.

## REFERENCES

1. Martin, W. B., and Essex, H. E.: Experimental Production and Closure of Atrial Septal Defects, With Observations of Physiologic Effects, Surgery 30: 283-297, 1951.
2. Little, R. C., Opdyke, D. F., and Hawley, J. G.: Dynamics of Experimental Atrial Septal Defects, Am. J. Physiol. 158: 241-250, 1949.
3. Dodrill, F. D.: A Method for Exposure of the Cardiac Septa: Experimental Study, J. THORACIC SURG. 18: 652-660, 1949.
4. Leeds, S. E.: Discussion of Swan, Maresh, Johnson, and Warner.[5]
5. Swan, H., Maresh, G., Johnson, M. E., and Warner, G.: The Experimental Creation and Closure of Auricular Septal Defects, J. THORACIC SURG. 20: 542-551, 1950.
6. Hanlon, C. R., and Blalock, A.: Complete Transposition of Aorta and the Pulmonary Artery: Experimental Observations on Venous Shunts as Corrective Procedures, Ann. Surg. 127: 385-397, 1948.
7. Kiriluk, L. B., Hoag, E. W., and Merendino, K. A.: Experimental Interauricular Septal Defects: A Physiologic Study With an Evaluation of Methods of Closure, Surg. Forum 1: 199-204, 1951.
8. Moore, T. C., and Shumacker, H. B., Jr.: Experimental Creation of Atrial Septal Defects, With Some Notes on the Production of a Right to Left Atrial Shunt, Angiology 4: 244-252, 1953.
9. Gross, R. E., Watkins, Elton, Jr., Pomeranz, A. A., and Goldsmith, E. I.: A Method for Surgical Closure of Interauricular Septal Defects, Surg., Gynec. & Obst. 96: 1-23, 1953.
10. Watkins, Elton, Jr., Pomeranz, A. A., and Gross, R. E.: Healing Patterns Following Closure of Experimental Defects of the Atrial Septum, Surg. Forum 4: 46-51, 1954.

than the figures given in Table IV. Similar estimates on the change in flow values showed a fall of 10 per cent in the magnitude of left-to-right shunt at three months, which was persistent at twelve months (Table V).

## COMMENT

When the techniques described in this paper were followed, satisfactory defects were produced which persisted in every animal. It is likely that some postoperative narrowing of these defects occurred, for the dimensions of the defects in animals which failed to survive the operation were greater than the dimensions of the defects in animals killed at intervals following operation. In all but one of the animals (No. 7) exactly the same surgical technique was used, so that it is likely that the difference between these two groups is the result of narrowing of the defect.

The preparation obtained cannot be regarded as simply an atrial septal defect, for the veins of the apical and cardiac lobes of the right lung now connect directly with the posterolateral margin of the right atrium proper. This does not invalidate considerations of the effects of such a defect relative to those of atrial septal defect seen in the human being.[23] Clinical cases of atrial septal defect have been seen in which a similar anomaly was found at operation. Furthermore, in the more usual type of atrial septal defect seen in the human patient, a greater proportion of blood from the right lung than from the left lung is shunted left to right.[24] The experimental preparation just described results in an anomaly the dynamic features of which closely simulate those in many cases of human atrial septal defect.

As indicated in an earlier section of this paper, an increased airway resistance produced by the recording spirometer for the measurement of oxygen uptake was considered likely. Calculation of the proportion of the left-to-right shunt while the animals breathed air indicated that the shunt was greater under this circumstance than while the animals breathed oxygen. This finding is contrary to observations made in many human patients in which oxygen has never been found to decrease the magnitude of the left-to-right shunt but usually causes a considerable increase. The data indicated that there is no progressive increase in the magnitude of the left-to-right shunt in the months following creation of the defect.

All the intracardiac pressures showed a moderate increase, which was usually present on the first study but was occasionally not seen until three months had elapsed. No further change in pressure appeared to occur beyond this time. As in human subjects with atrial septal defects, a difference in pressures has been frequently noted between the main pulmonary artery and the chamber of the right ventricle. In most instances this gradient of systolic pressure was noted when the catheter tip was moved from the outflow tract of the right ventricle to the region just within the tricuspid valve. In a few instances the pressure change occurred at the pulmonary valve itself. This variation in systolic pressure within the right ventricle has also been described in dogs with ventricular septal defect by King and associates[25] with findings similar

# WAR WOUNDS OF THE HEART

## REPORT OF TWO PATIENTS WITH UNUSUAL FEATURES

FRANK C. SPENCER, M.D., AND JOHN H. KENNEDY, M.D.
BALTIMORE, MD.

### INTRODUCTION

ARMORED vests worn by combat personnel in the Korean War were highly effective in preventing injury from low velocity missiles. The 2 patients discussed in this report had the only cardiac wounds seen in an unselected group of 2,000 battle casualties treated at a forward surgical hospital in Korea. Both patients were Koreans, and were not wearing armored vests. One patient was a problem in the diagnosis and treatment of acute cardiac tamponade, cardiac hemorrhage, cardiac arrest, and embolization of an intracardiac foreign body to a peripheral artery. The other patient had a cardiac tamponade from wounds of both ventricles.

The largest series of cardiac injuries in World War II was reported by Samson,[1] who described experiences with 57 cardiac and 18 pericardial wounds. The cardiac injuries included 26 myocardial contusions, 10 lacerations, 19 perforations, and 2 wounds associated with embolization of the missile. The symptoms in most cases were those of shock. Cardiac tamponade was present in only 5 patients. Chest roentgenograms often did not show a small intracardiac foreign body because of the density of the heart shadow. A diagnosis of a cardiac wound was not made in 15 of the 57 patients before autopsy. Many of the cardiac wounds in the other 47 patients were first recognized during a thoracotomy performed because of the associated chest injury. A total of 29 cases of myocardial lacerations and perforations were found at thoracotomy. Sixteen of these wounds were sutured, but 11 did not require any sutures; 2 patients died of uncontrollable hemorrhage. The prognosis was most closely related to the extent of the myocardial contusion. Twenty deaths occurred in the 57 patients with cardiac wounds; 11 of these were from the extent of the myocardial injury, 4 from hemorrhage, and 2 from tamponade. The prognosis after the first postoperative day was favorable, for only 3 of the 20 deaths occurred after this time. Hillsman[2] reported 2 survivors in a group of 4 patients with cardiac wounds in whom a myocardial laceration was sutured. Shock was present in each patient, but tamponade was not observed.

Cardiac wounds in civilian practice have been reported by several authors. The largest groups of patients reported are tabulated in Table I. The injuries

---

From the Department of Surgery, The Johns Hopkins Hospital and The Johns Hopkins University School of Medicine, Baltimore, Md.

Received for publication Feb 27, 1956.

11. Donald, D. E., Kirklin, J. W., Ellis, F. H., Jr., and Grindlay, J. H.: Methods of Closure of Experimentally Produced Atrial Septal Defects, Surg. Forum 4: 41-46, 1954.
12. Silver, A. W., Swan, H. J. C., and Kirklin, J. W.: Demonstration by Dye Dilution Technics of Preferential Flow Across Atrial Septal Defects From Right Pulmonary Veins and Inferior Vena Cava (Abstr.), Fed. Proc. 13: 138, 1954.
13. Griffin, G. D. J., and Essex, H. E.: Experimental Production of Interventricular Septal Defects: Certain Physiologic and Pathologic Effects, Surg., Gynec. & Obst. 92: 325-332, 1951.
14. Miller, M. E.: Guide to the Dissection of the Dog, ed. 3, Ann Arbor, Mich., 1952, Edwards Bros., Inc., p. 158.
15. Barclay, A. E., Franklin, K. J., and Prichard, M. M. L.: The Foetal Circulation, Oxford, 1944, Blackwell Scientific Publications, Ltd., p. 227.
16. Wood, E. H.: Special Technics of Value in the Cardiac Catheterization Laboratory, Proc. Staff Meet., Mayo Clin. 28: 58-64, 1953.
17. Wood, E. H.: Oximetry. In Glasser, Otto: Medical Physics, Chicago, 1950, The Year Book Publishers, Inc., vol. 2, pp. 664-680.
18. Van Slyke, D. D., and Neill, J. M.: The Determinations of Gases in Blood and Other Solutions by Vacuum Extraction and Manometric Measurement. I., J. Biol. Chem. 61: 523-573, 1924.
19. Pappenheimer, J. R., and Associates: Standardization of Definitions and Symbols in Respiratory Physiology, Fed. Proc. 9: 602-605, 1950.
20. Sendroy, J., Jr.: Manometric Determination of Hemoglobin by the Oxygen Capacity Method, J. Biol. Chem. 91: 307-323, 1931.
21. Wood, E. H., and Cronin, L.: Normal Oxygen Saturation of Arterial Blood During Inhalation of Air and Oxygen, J. Appl. Physiol. 1: 567-574, 1949.
22. Swan, H. J. C., and Wood, E. H.: Localization of Cardiac Defects by Dye-dilution Curves Recorded After Injection of T-1824 at Multiple Sites in the Heart and Great Vessels During Cardiac Catheterization, Proc. Staff Meet., Mayo Clin. 28: 95-100, 1953.
23. Kirklin, J. W., Swan, H. J. C., Wood, E. H., Burchell, H. B., and Edwards, J. E.: Anatomic, Physiologic, and Surgical Considerations in Repair of Interatrial Communications in Man, J. THORACIC SURG. 29: 37-49, 1955.
24. Swan, H. J. C., Hetzel, P. S., and Wood, E. H.: Quantitative Estimation by Indicator-Dilution Technics of Contribution of Blood From Each Lung to the Left-to-Right Shunt in Atrial Septal Defect, Circulation. In press.
25. King, H., Coggeshall, W. E., Lurie, P. R., and Shumacker, H. B., Jr.: Cardiodynamics of Experimental Ventricular Septal Defects in Dogs, Surg. Forum 4: 115-122, 1954.
26. Blount, S. G., Jr., Swan, H., Gensini, G., and McCord, M. C.: Atrial Septal Defect: Clinical and Physiologic Response to Complete Closure in Five Patients, Circulation 9: 801-812, 1954.
27. Haddy, F. J., Alden, J. F., Ferrin, A. L., Hannon, D. W., Adams, W. L., and Baronofsky, I. D.: An Evaluation of Wedge Pressures in Dogs Under Conditions of Normal and Elevated Pulmonary Vascular Pressures, Circulation Res. 1: 157-161, 1953.
28. Connolly, D. C., Kirklin, J. W., and Wood, E. H.: The Relationship Between Pulmonary Artery Wedge Pressure and Left Atrial Pressure in Man, Circulation Res. 2: 434-440, 1954.

Anesthesia was begun with Pentothal and succinylcholine, and maintained with ether and nitrous oxide. The left chest was opened through an incision in the fifth interspace anteriorly. There was a total of about 500 ml. of blood in the pleural cavity. The pericardium was distended with blood. Two small lacerations on the anterior surface of the pericardium were sealed by blood clots. The pericardium was opened, and about 200 ml. of tightly packed blood clots removed. Two myocardial lacerations were present, each about 1 cm. in diameter. One was in the anterior wall of the right ventricle, and the other in the wall of the left ventricle near the apex. The right ventricular wound was bleeding slowly. The left ventricular wound spurted blood with each systolic contraction. Each wound was repaired with interrupted sutures of medium silk tied over a strip of Gelfoam. The pericardium was partly closed, and the chest wall was closed in layers with interrupted silk sutures. An intercostal drainage catheter was placed posteriorly in the seventh interspace. The multiple small wounds of the extremities were débrided and packed with gauze. The patient received 1,500 ml. of blood during the operation. At the end of the operation the blood pressure was 100/60 mm. Hg.

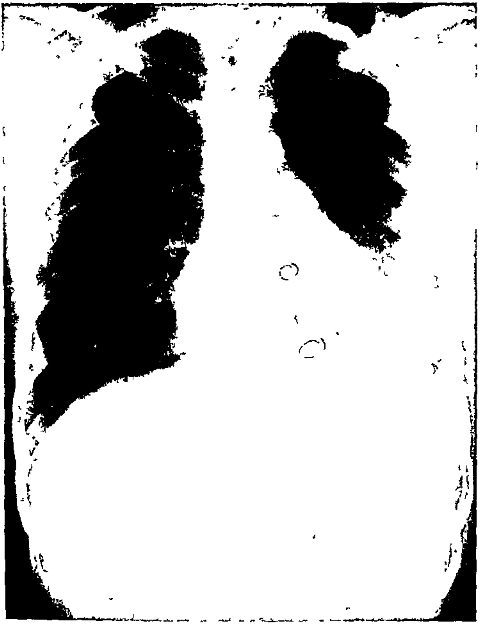


Fig. 2.—Chest roentgenogram of Case 2, posteroanterior view. Two small mortar fragments are barely visible within the cardiac shadow.

The patient had an uneventful postoperative course. Chemotherapy included penicillin, streptomycin, and tetanus toxoid. A low grade fever subsided by the tenth postoperative day. The urinary output was normal. An electrocardiogram on the second postoperative day was interpreted as showing evidence of diffuse myocardial injury. Subsequent electrocardiograms showed a gradual return to normal. The foreign bodies were localized by lower lobe of the lung was diffusely hemorrhagic, but no laceration was identified. No other wound of the heart could be found. The pericardium was partly closed, and the chest wall closed in layers with interrupted silk sutures. The pleural cavity was drained with tubes placed through intercostal stab wounds. The abdominal incision was closed with a single row of interrupted figure-of-eight steel wire sutures. The missile wounds in the chest wall were débrided and packed loosely with gauze. Two thousand milliliters of blood were given during the operation, which lasted two hours.

The postoperative treatment included the administration of penicillin, streptomycin, and tetanus toxoid, and bed rest. The patient was awake two hours after operation and showed no abnormal neurologic signs. A chest roentgenogram the next day showed complete expansion of the lungs and two 1.5 cm. metallic foreign bodies. One was located at the periphery of the right lung field at the level of the second rib posteriorly, and the other was in the periphery of the left lung field at the level of the tenth rib. The chest roentgenogram is reproduced in Fig. 1. No pulse or blood pressure could be obtained in the right arm, but there was no evidence of circulatory insufficiency. The urinary output was normal.

The patient had a persistent fever in the early postoperative course, with daily elevations of 102° to 104° F. Daily red blood cell counts showed a progressive decrease from 5.7 million cells per cubic millimeter on the first postoperative day to 2.7 million on the fifth postoperative day, at which time the hematocrit was 27 per cent. The white blood cell counts were 13,000 to 16,000 cells per cubic millimeter. Blood transfusions of 500 ml. each were given on the sixth and seventh postoperative days. The fever, anemia, absent right radial pulse, and location of the foreign body in the right supraclavicular area led to a diagnosis of foreign body embolism from the left ventricle to the right subclavian artery, with a secondary septic endarteritis. No blood cultures were obtained because of the absence of bacteriologic equipment. Penicillin therapy was increased to one million units intramuscularly every 3 hours on the twelfth postoperative day, and the patient became afebrile four days later. The surgical incisions healed well, and the missile wounds healed following secondary suture. Electrocardiograms in the early postoperative course were interpreted as showing an area of injury in the left ventricle. Electrocardiograms taken several weeks after injury showed almost complete disappearance of the abnormalities seen earlier.

The patient was evacuated to a hospital ship on the eighteenth postoperative day. The missile was removed from the subclavian artery on the twenty-second postoperative day, at which time it was found surrounded by a 3 by 2 cm. inflammatory mass containing purulent fluid. The subclavian artery was ligated. A chest roentgenogram one month following injury showed a normal cardiac shadow. The patient was asymptomatic ten weeks after injury and had a normal exercise tolerance. There were no cardiac murmurs. The right radial pulse was absent, but there were no signs of circulatory insufficiency.

Case 2.—The patient, C. K. Y., was a Korean marine, age 25, admitted from helicopter evacuation on April 27, 1953, ninety minutes after receiving multiple wounds from the explosion of a land mine. The patient was restless but rational. The radial pulse was barely palpable; no blood pressure could be obtained. There were two irregular 1 cm. wounds in the anterior chest wall, one in the midline over the upper sternum and the other in the fourth interspace at the left sternal border. The neck veins were greatly engorged when the patient was in the recumbent position. The breath sounds were decreased over the left chest; the heart sounds could not be heard. There were small wounds on both arms, both legs, and the left foot. A total of 3,000 ml. of type "O" blood was given in forty minutes wtihout any rise in blood pressure. A thoracentesis in the left chest did not produce any air or fluid. A chest roentgenogram showed an enlarged, globular cardiac shadow with two irregular metallic foreign bodies, each about 4 mm. in diameter, within the cardiac shadow. Pericardial aspiration in the left fifth interspace removed 40 ml. of blood. There was an immediate rise in blood pressure to 90/60 mm. Hg, and a definite improvement in the patient's general condition. He was then taken to the operating room for thoracotomy.

and rigidity, and the patient's restlessness and shouting which made auscultation of the chest difficult. Pericardial aspiration was of immediate diagnostic and therapeutic benefit in Case 2, and would probably have been similarly useful in Case 1. Repeated pericardial aspirations might have avoided a thoracotomy in Case 2, although the left ventricular wound was actively bleeding when exposed at thoracotomy.

Pericardial aspiration has been very successful in the nonoperative treatment of heart wounds seen in civilian practice. Following the use of this method of therapy by Blalock and Ravitch,[14, 8] good results have been obtained by aspiration in large groups of cases by Elkin,[9] Carr,[12] and Cooley,[11] and their associates, and Griswold and Drye.[13] Naclerio and co-workers[10] have continued to treat all cardiac wounds with immediate operation and suture of the myocardial laceration.

*Cardiac Arrest.*—The prompt appearance of cardiac contractions following cardiac massage in Case 1 may be attributed to the short time interval between the onset of cardiac arrest and the beginning of cardiac massage. Survival after cardiac arrest due to cardiac tamponade is rare. A patient reported by Loken[15] died from neurologic injuries thirty days after resuscitation from cardiac arrest by cardiac massage. The cardiac arrest occurred during the induction of anesthesia. No other patient was found in the literature reviewed in whom cardiac activity was restored after cardiac tamponade had resulted in cardiac arrest.

*Migration of Missiles.*—Intravascular migration of a foreign body is an infrequent occurrence. Reviews of the reported cases have been published by Movin,[16] Harken and their associates,[18, 19] Keeley,[17] Straus,[20] and Decker.[21] These reviews contain a total of 21 patients in whom a foreign body entered the chest and embolized to a peripheral artery. Nine patients survived; 4 of the patients had an amputation performed because of gangrene in the involved extremity. Samson,[1] Griswold and Maguire,[22] and Dickinson[23] each had one patient who is not included in the above-mentioned reviews. The patients of Samson and Griswold are similar in that both patients had no symptoms from a bullet that had entered the chest and lodged in the region of the iliac artery; the bullets were not removed. Dickinson's patient had the sudden onset of pain and paralysis in the left leg several months after recovery from a bullet wound of the chest. Full recovery followed removal of the bullet from the left external iliac artery. This patient is the only one in whom several months elapsed between injury and embolism of the missile.

The addition of Case 1 in this report makes a total of 25 patients with thoracic injuries and subsequent embolization of the missile to a peripheral artery. Most of the missiles embolized to the arteries of the lower extremities. The missile lodged in the femoral artery in 11 patients, the iliac artery in 7, the abdominal aorta in 2, and the popliteal artery in one patient. The 3 missiles embolizing to the upper extremity lodged in the innominate artery, the subclavian artery, and the axillary artery in one patient each. In the patients reviewed by Keeley,[17] the embolus was in the right iliac or femoral fluoroscopy in the region of the left auricle and the ventricle. The chest roentgenograms ar reproduced in Figs. 2 and 3. Surgical removal of the foreign bodies was not felt to b indicated. A Grade 2 harsh systolic murmur was heard over the base of the heart in th early postoperative period. The murmur gradually decreased in intensity and disappeare after the sixth postoperative week. The patient was asymptomatic when last seen ten week after injury.

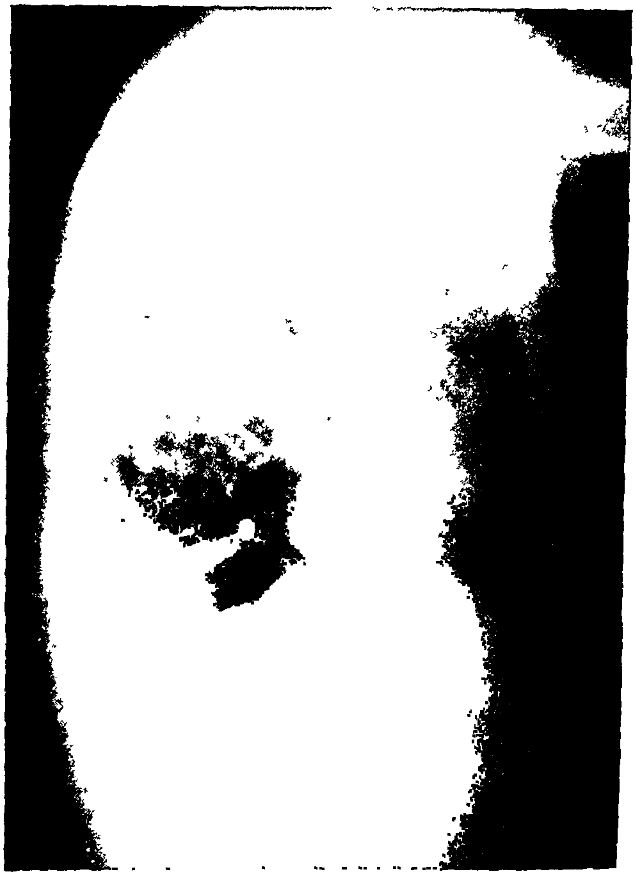


Fig. 3.—Chest roentgenogram of Case 2, lateral view. The 2 small mortar fragments are visible in the region of the left auricle and left ventricle.

## DISCUSSION

A striking feature in each patient was the extreme restlessness in the presence of severe shock. Such restlessness was seldom seen in patients in shock from other causes. A possible explanation for the restlessness could be that the cerebral anoxia is more severe in shock from cardiac tamponade than in shock from other causes. The combination of arterial hypotension and venous hypertension may decrease cerebral blood flow more than just arterial hypotension alone. Another noteworthy feature in both cases was the failure of shock to respond to the rapid intravenous administration of blood. These two observations in a person with a chest wound should suggest acute cardiac tamponade. The diagnosis can be confirmed by pericardial aspiration. The error in diagnosis in Case 1 was caused by the location of the wounds which suggested a perforating thoracico-abdominal wound, the abdominal distention group of 2,000 battle casualties. This low incidence is attributed to the wearing of armored vests by combat personnel. Cardiac tamponade was present in each patient. A cardiac injury in one patient was recognized only after cardiac arrest terminated an emergency laparotomy begun because of an error in diagnosis. Cardiac function was restored by correction of the cardiac tamponade and cardiac massage. A 2 cm. perforation in the wall of the left ventricle was then sutured. The mortar fragment embolized to the right subclavian artery and caused a septic endarteritis. A good result was obtained by removal of the foreign body and ligation of the subclavian artery.

Twelve other patients were found in the surgical literature who survived a wound produced by a missile that entered the chest and subsequently embolized to a peripheral artery. Most of the missiles lodged in arteries of the lower extremities. Gangrene developed in the involved extremity in 4 patients. No patient was found in the surgical literature who survived cardiac arrest resulting from cardiac tamponade.

The other patient had cardiac tamponade from lacerations of the right and left ventricles from two small mortar fragments. A good result was obtained by pericardial aspiration and subsequent suture of the myocardial lacerations. Removal of the intracardiac foreign bodies was not felt to be indicated.

## REFERENCES

1. Samson, P. C.: Battle Wounds and Injuries of the Heart and Pericardium, Ann. Surg. 127: 1127-1149, 1948.
2. Hillsman, J. A. B.: Some Observations on Penetrating Wounds of the Heart, Am. J. Surg. 73: 305-312, 1947.
2. Bigger, I. A.: The Diagnosis and Treatment of Heart Wounds, South. M. J. 33: 6-11, 1940.
4. Nelson, H.: Penetrating Wounds of the Heart, Arch. Surg. 47: 571-582, 1943.
5. Linder, H. and Hodo, H.: Stab Wounds of the Heart and Pericardium, South. M. J. 37: 261-266, 1944.
6. Blau, M. H.: Wounds of the Heart, Am. J. M. Sc. 210: 252-262, 1945.
7. Maguire, C. G., and Griswold, R. A.: Further Observations on Penetrating Wounds of the Heart and Pericardium, Am. J. Surg. 74: 721-731, 1947.
8. Ravitch, M. M., and Blalock, A.: Aspiration of Blood From Pericardium in Treatment of Acute Cardiac Tamponade After Injury, Arch. Surg. 58: 463-477, 1949.
9. Elkin, D. C., and Campbell, R. E.: Cardiac Tamponade: Treatment by Aspiration, Ann. Surg. 133: 623-627, 1951.
10. Maynard, A. D. L., Cordice, J. W. V., and Naclerio, E. A.: Penetrating Wounds of the Heart, Surg., Gynec. & Obst. 94: 605-618, 1952.
11. Cooley, D. A., Dunn, J. R., Brockman, H. L., and DeBakey, M. E.: Treatment of Penetrating Wounds of the Heart: Experimental and Clinical Observations, Surgery 37: 882-889, 1955.
12. Farringer, J. L., and Carr, D.: Cardiac Tamponade, Ann. Surg. 141: 437-442, 1955.
13. Griswold, R. A., and Drye, J. C.: Cardiac Wounds, Ann. Surg. 139: 783-785, 1954.
14. Blalock, A., and Ravitch, M. M.: A Consideration of the Nonoperative Treatment of Cardiac Tamponade Resulting from Wounds of the Heart, Surgery 14: 157-162, 1943.
15. Loken, A. C., Haymaker, W., and Paulson, D. L.: Stab Wound of the Heart Followed by Temporary Cessation of the Heartbeat With Resuscitation by Cardiac Massage, Surgery 26: 745-755, 1949.
16. Movin, R., Russell, J., and Valle, A. R.: A Migratory Arterial Foreign Body, Am. J. Surg. 91: 118-120, 1956.
17. Keeley, J. L.: A Bullet Embolus to the Left Femoral Artery Following a Thoracic Gunshot Wound, J. THORACIC SURG. 21: 608-620, 1951.
18. Harken, D. E.: Foreign Bodies In, and In Relation To, the Thoracic Blood Vessels and Heart, Surg., Gynec. & Obst. 83: 117-125, 1946.
19. Harken, D. E., and Williams, A. C.: Foreign Bodies In and In Relation to the Thoracic Blood Vessels and Heart, Am. J. Surg. 72: 80-90, 1946.

artery in 2 patients whereas it was in the left femoral or iliac artery in 14 patients. The predominance of left-sided embolization was attributed to the fact that the junction between the left iliac artery and aorta is a smaller angle than the junction between the right iliac artery and aorta.

The missile was removed from the artery in all but 2 of the 13 patients who survived the injury. The usual indication for removal was circulatory insufficiency of the extremity. Amputations for gangrene were subsequently performed in 4 patients. The time interval between embolization and removal of the embolus was greater than seventeen hours in 3 of the 4 cases, and was not stated in the fourth case. Case 1 in this report is the only patient in whom a septic endarteritis developed from the missile.

The review by Harken and Williams[19] includes patients with missiles migrating within the venous circulation. The route of embolism varied in different patients, but in a total of 9 patients the missile entered a large vein, embolized to the right ventricle, and remained in this location.

*Intracardiac Foreign Bodies.*—The removal of the 2 small foreign bodies in Case 2 was not considered necessary. Fluoroscopic examination showed the missiles to have an intracardiac location, but fluoroscopic localization is subject to considerable error. One third of a large group of patients operated on by Harken[18] were found on surgical exploration to have the missiles in an extracardiac location, although preoperative fluoroscopy had indicated an intracardiac location. The clinical indications for removal of intracardiac foreign bodies have been discussed by Swan and his colleagues,[24] Harken,[18] and Decker.[21] Recurrent pericardial effusion or signs of infection have been the usual indications for removal. The development of these complications from a foreign body may depend upon the development of infection, for experimental studies have shown that sterile foreign bodies are well tolerated in various locations in the heart.[24, 25] Large foreign bodies are more commonly associated with an infection than small ones, perhaps because fragments of clothing often adhere to large foreign bodies.

The surgical results from removal of foreign bodies have been good. Swan[24] reported that a total of 27 patients have had excision of a foreign body from an intracardiac location, and over 150 patients have had missiles removed from areas adjacent to the heart.[18, 26] There has been no mortality in either group of patients.

*Prognosis.*—No limitation of cardiac function is expected in either patient reported. Myocardial failure has not been reported in follow-up studies of patients recovering from cardiac wounds. Pericardial adhesions have rarely caused any disability. A collected series of patients with constrictive pericarditis appearing after recovery from cardiac wounds has been discussed recently by McKusick, Kay, and Isaacs.[27]

## SUMMARY

Two patients with penetrating wounds of the heart from mortar fragments are reported. These patients had the only cardiac injuries seen in an unselected

# LIPOMA OF THE BRONCHUS

## Report of a Case

Seymour Ochsner, M.D., Francis E. LeJeune, M.D., and Alton Ochsner, M.D.
New Orleans, La.

INTEREST in benign bronchial tumors and increasing appreciation of their importance have paralleled the development of bronchoscopic and radiologic methods of bronchial examination. Improved methods of endoscopic and thoracic surgery now permit successful management of most bronchial tumors. Statistical reports of large series of bronchial neoplasms suggest that benign tumors represent 5 to 10 per cent of the total.[3, 7, 11] Among these benign tumors is the bronchial lipoma, which is sufficiently rare to warrant report of a case. This case is, we believe, the fourteenth to be recorded. It clearly illustrates some of the problems associated with management of benign bronchial tumors in general.

The 3 cases of benign bronchial tumors reported before 1927, which were apparently lipomas or similar tumors with a large fatty component, were discovered at autopsy and were of undetermined clinical significance (Table I). In 1927, Kernan[6] reported the first case of bronchial lipoma seen at bronchoscopy and successfully removed through the bronchoscope. As indicated in Table I, other reported lipomas were so situated as not to be removable bronchoscopically, or resulted in bronchial obstruction, bronchiectasis, or such secondary pulmonary damage that pulmonary resection was necessary.

### CLINICAL MANIFESTATIONS

In lipomas of the bronchus, as in other benign bronchial tumors, the symptoms and signs are largely dependent on the location of the tumor and the degree of bronchial obstruction produced.[17] The study of Watts and co-workers[16] indicated that bronchial lipomas undoubtedly arise from fatty tissue normally present in the bronchial tree, where it occurs in all bronchi which have cartilaginous plates in their wall (i.e., those bronchi 1 mm. or larger in diameter). Although more fatty tissue is found in the bronchial wall external to the cartilaginous plates, it seems that submucosal lipomas (those arising internal to the cartilaginous plates) are more significant clinically because they expand endobronchially and produce bronchial obstruction more readily.

Cough, resulting from bronchial irritation, may be the earliest symptom. Unlike the highly vascular adenomas, which bleed readily,[3, 17] lipomas are

---

From the Departments of Radiology, Otorhinolaryngology, and Surgery, Ochsner Clinic, New Orleans, La.

Received for publication March 7, 1956.

20. Straus, R.: Pulmonary Embolism Caused by a Lead Bullet Following a Gunshot Wound of the Abdomen, Arch. Path. 33: 63-68, 1942.
21. Decker, H. R.: Foreign Bodies in the Heart and Pericardium—Should They Be Removed? J. THORACIC SURG. 9: 62-79, 1939.
22. Griswold, R. A., and Maguire, C. H.: Penetrating Wounds of the Heart and Pericardium, Surg. Gynec & Obst. 74: 406-418, 1942.
23. Dickinson, E. H.: Personal communication.
24. Swan, H., Forsee, J. H., and Goyette, E. M.: Foreign Bodies in the Heart, Ann. Surg. 135: 314-323, 1952.
25. Fritz, J. M., Newman, M. N., Jampolis, R. W., and Adams, W. E.: Fate of Cardiac Foreign Bodies, Surgery 25: 869-879, 1949.
26. Miscall, L., and Harrison, A. W.: Thoracic Surgery in a Hospital Center, Ann. Surg. 125: 311-333, 1947.
27. McKusick, V. A., Kay, J. H., and Isaacs, J. P.: Constrictive Pericarditis Following Traumatic Hemopericardium, Ann. Surg. 142: 97-103, 1955.

relatively avascular and less frequently produce hemoptysis. As the tumor expands into the bronchial lumen, it interferes with the airway and may cause wheezing. If air is trapped in the pulmonary segment, localized obstructive emphysema may result. As the obstruction becomes complete, atelectasis occurs in the involved pulmonary segments or lobes. Bronchial secretions are trapped in the occluded bronchi; pneumonitis and bronchiectasis follow. Extensive pulmonary damage ultimately results if the bronchial obstruction is not relieved. The clinical course of many patients with benign bronchial tumors is featured by a history of recurring episodes of productive cough, pulmonary symptoms, and "pneumonia" for several years. These occur during the period when the slowly growing tumor is large enough to produce intermittent bronchial obstruction. Recurring episodes of "pneumonia" demand complete investigation of the bronchial tree.

## ROENTGENOLOGIC MANIFESTATIONS

Since a lipoma is more apt to occur in a large bronchus than in a small one, the tumor is usually located in the hilar region of the lung. A hilar neoplasm may first become evident in a roentgenogram of the chest as an unusually prominent hilar shadow. The tumor may give the hilum an unusual density, a broader or longer shadow than normal, or an eccentric or unusual configuration. In the earliest stages of the tumor's growth, this manifestation may escape detection. Careful comparison of serial roentgenograms should give a clue to the presence of disease, however, as benign neoplasms gradually increase in size.

Endobronchial growth of a tumor can usually be demonstrated by bronchographic study before it is manifest in plain roentgenograms. Thorough bronchographic exploration of the bronchial tree, as pointed out by Di Rienzo,[1] will disclose small intraluminal lesions. A polypoid growth stands out as a rounded, oval, or irregular filling defect in the bronchus, which has been outlined by contrast media coating its mucosal surface.

Stenosing bronchial growths will be portrayed as smooth or irregular narrowing of the bronchial lumen. Above the lesion there may be some apparent enlargement of the bronchus, perhaps associated with overfilling or slow emptying of this segment. The stenosis may be incomplete or complete. If incomplete, the bronchi below the narrowing may be fragmentarily visualized, may be partially filled with retained secretions, or may show varying degrees of bronchiectasis. If the obstruction is complete, no contrast media will pass into the bronchial segments peripheral to the tumor. The occluding tumor may be indicated by a rounded endobronchial mass outlined by contrast media, or at times by a soft tissue extrabronchial mass at the site of the obstruction.

After the tumor has produced bronchial obstruction, resulting changes in the lung are manifest in plain roentgenograms. Localized obstructive emphysema may be the first indication of an endobronchial growth. This occurs because the bronchial lumen enlarges during the inspiratory phase of respiration and becomes smaller during expiration. Therefore, the involved

TABLE I. SUMMARY OF CASES OF LIPOMA OF THE BRONCHUS COLLECTED FROM THE LITERATURE

| AUTHOR | YEAR | AGE, SEX, SYMPTOMS | ROENTGENOLOGIC DATA | BRONCHOSCOPY | TREATMENT | COMMENTS |
|---|---|---|---|---|---|---|
| Rokitansky[12] | 1854 | | | | | Autopsy finding, mentioned occurrence only |
| Laboulbene[8] | 1879 | | | | | Autopsy finding; spindle and fat cells |
| Feller[2] | 1922 | 66, F | | Not done | | Autopsy finding, RML; lipoma, or hamartoma |
| Kernan[6] | 1927 | 50, M | | Tumor seen | Bronchoscopic removal | |
| Myerson[10] | 1928 | 47, F | | Tumor seen | Bronchoscopic removal | Fibrolipoma, improved |
| Jackson and Jackson[5] | 1932 | 64, productive cough, hemoptysis | | Tumor seen | Bronchoscopic removal | Symptoms disappeared |
| | | No data | | Tumor seen | Bronchoscopic removal | Fibrolipoma; ? tracheal or bronchial |
| Wessler and Rabin[17] | 1932 | 48, M, thoracic pain, productive cough, dyspnea, hemoptysis | Infiltration, LLL | Tumor seen | Bronchoscopic removal | Improved |
| Honig[4] | 1934 | 54, M | | Not done | | Autopsy finding; left main bronchus |
| McGlade[9] | 1939 | 48, M, productive cough, wheeze, recurrent pneumonia | Atelectasis, LLL | Tumor seen | Bronchoscopic removal | Fibrolipoma, improved but bronchiectasis persisted |
| Vinson and Pembleton[15] | 1942 | 29, F, productive cough, fever | Atelectasis and abscesses, left lung | Tumor seen | Bronchoscopic coagulation | Improved, but lung suppuration persisted |
| Watts and associates[16] | 1946 | 51, F, fatigue, fever, weight loss, cough | Hilar mass, bronchial obstruction, infiltration, LUL | Not done | Lobectomy, LUL, 2 cm. tumor, bronchiectasis | Recovery |
| Whalen[18] | 1947 | 61, M, chills, fever, thoracic pain | Infiltration and atelectasis, left lung | Tumor seen | Bronchoscopic removal | Improved but bronchiectasis persisted |
| Touroff and Seley[14] | 1951 | 48, F, cough, hemoptysis | Subhilar mass, shrunken LLL | Not done | Lobectomy, LLL | Recovery |

being necessary[14] The need for prompt therapy is great and there should be no hesitancy to perform exploratory thoracotomy if the endoscopist is unable to remove the lesion completely, or if subsequent studies reveal significant bronchiectasis or bronchostenosis

## CASE REPORT

C T, a 35 year old machinist, was well until the winter of 1943 when some wheezing, slight pain in the midsternal area, generalized aching, and mild fever developed These symptoms disappeared following treatment by his local physician with a sulfonamide The sense of retrosternal oppression returned, however A roentgenogram of the chest showed a "spot" in the lung Bronchoscopic examination revealed a mass in the right bronchus from which a specimen was obtained He was told he had a tumor which would be removed bronchoscopically However, four bronchoscopic attempts to remove the tumor proved unsuccessful He was then advised that operative removal would be necessary

When first seen at the Ochsner Clinic on Jan 26, 1945, the patient was asymptomatic It had been three months since the last bronchoscopic examination During this interval he had occasionally noticed transitory inability to take a deep breath The only attacks of hemoptysis he had had followed each bronchoscopic attempt at removal of the tumor The patient had lost no weight, and had not had a productive cough or dyspnea

On admission, slight decrease in resonance was noted on percussion of the right anterior chest A roentgenogram of the chest on the day of admission showed nodular prominence of the right hilum, with linear shadows extending into the peripheral right pulmonary field (Fig 1) There was some increased density at the right cardiophrenic angle with a slight shift of the trachea to the right, suggestive of the presence of partial atelectasis of a portion of the right lung The right main bronchus was visualized, but the intermediate bronchus was not delineated

Bronchography, done the following day, indicated occlusion of the right middle lobe bronchus near its origin, with resulting atelectasis of the middle lobe (Fig 2) Irregular dilatation of some of the bronchi of the anterior segment of the upper lobe and compensatory overexpansion of the right lower lobe were apparent

Bronchoscopy under local anesthesia on Feb 26, 1945, disclosed a smoothly rounded, firm tumor which seemed to fill the right bronchus completely On the first two attempts at removing the tumor, a small portion broke off On the third attempt, the entire tumor seemed to come up through the bronchoscope The yellowish white tumor was 3 cm long and 5 mm in diameter (Fig 3) Microscopic examination proved the lesion to be a submucosal lipoma of the bronchus, with areas of fat necrosis, chronic inflammation, and fibrosis

Bronchograms made two days after removal of the obstructing lesion showed that it had been removed from the right middle lobe bronchus The middle lobe bronchial segments, outlined by Lipiodol, showed severe bronchiectasis (Fig 4) The middle lobe was considerably shrunken The localized bronchiectasis in the inferior portion of the anterior segment of the right upper lobe persisted

The patient was discharged in the hope that the bronchiectasis would subside after removal of the obstructing lipoma However, a bronchogram on April 12, 1945 indicated persistence of the bronchiectasis The patient refused to submit to operation On Sept 24, 1945, a bronchogram showed no Lipiodol passing into the middle lobe bronchus Whether this obstruction was due to secretions, a plug of mucus or regrowth of the tumor was undetermined The patient refused bronchoscopic examination but agreed to surgical intervention

On Oct 1, 1945, resection of a shrunken middle lobe, along with wedge resection of the anteroinferior portion of the right upper lobe was carried out Pathologic examination showed severe bronchiectasis and chronic bronchitis There was no evidence of residual or recurrent lipoma Postoperative recovery was prompt and uncomplicated segment is overinflated as a result of air being trapped within it. In roentgenograms this may be seen as an unusually radiolucent segment or lobe in the pulmonary field. It is most noticeable if roentgenograms are obtained at two phases of the respiratory cycle, with the patient in full inspiration and complete expiration. Careful fluoroscopy of the chest is another valuable aid in determining the presence of localized obstructive emphysema.

When the tumor completely fills the bronchial airway, the air trapped in the lung distal to the obstruction is absorbed and atelectasis ensues. This is evident in roentgenograms as a relatively homogeneous shadow involving a segmental or lobar area of the lung. If the pulmonary area involved is sizable, other portions of the lung may show overexpansion, or there may be other compensatory changes, as mediastinal shift or elevated diaphragm.[13]

## BRONCHOSCOPIC MANIFESTATIONS

Development of endoscopic methods made possible direct inspection of the trachea and large bronchi. Bronchoscopic study is a valuable feature in both the diagnosis and treatment of benign bronchial neoplasms. The position, size, and gross characteristics of the tumor, the mucosa over the tumor and in the adjacent bronchus, and the degree of attendant bronchial obstruction, can be determined bronchoscopically. Endoscopic biopsy often establishes a histologic diagnosis. A bronchial lipoma usually appears as a smooth, rounded, or oval, submucosal mass. It is firm and gray or yellowish in color; the overlying mucosa is stretched but rarely ulcerated. The bronchial lumen shows various degrees of obstruction. Pus may be seen exuding around the mass from the bronchus beyond it.[15] If the tumor is partially or completely fulgurated or excised, bronchiectasis may be evident in the bronchial segments distal to it.

## TREATMENT

Benign bronchial tumors demand prompt excision. As Myerson[10] wrote, they are "benign only in the histologic sense." The damage they produce, when untreated, may prove fatal.

When seen by the endoscopist, the tumor may be fulgurated or excised by bronchoscopic means. Apparent successful removal of lipomas has been reported in 7 instances (Table I), and was accomplished also in the case to be reported. This method is not without some danger of complications. Mediastinal emphysema, tracheobronchial hemorrhage, and death from cardiac trauma have been reported as complications following bronchoscopic removal of bronchial tumors.[11, 18]

In submucosal lipomas of the bronchus, the chief disadvantage of bronchoscopic removal seems to be the persistence of bronchiectasis after the tumor has been removed. Three such instances have been reported (Table I). In the case to be presented subsequent pulmonary excision was required to cure the severe localized bronchiectasis.

Lipomas of the upper lobe bronchi may not be visible to the bronchoscopist, or may not be accessible for endoscopic therapy. Tumors with extrabronchial extension cannot be managed bronchoscopically, primary transthoracic excision

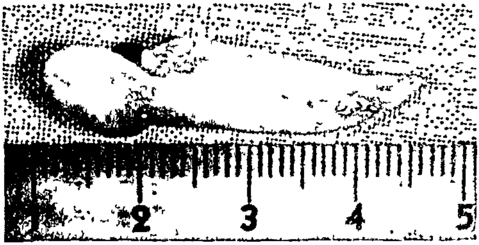


Fig 3 —Photograph of submucosal lipoma of the bronchus following bronchoscopic removal

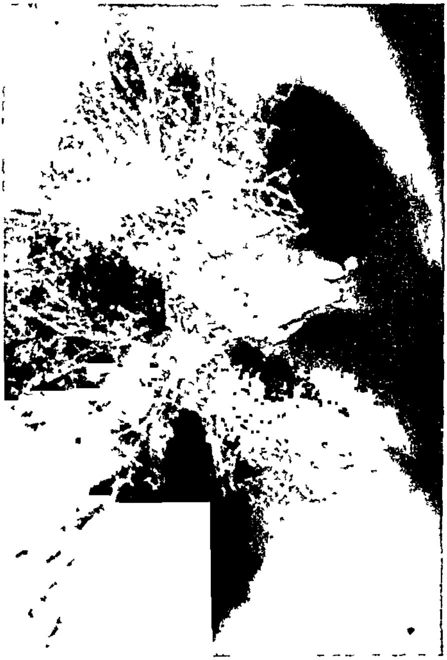


Fig 4 —Right lateral bronchogram after bronchoscopic removal of lipoma Partially collapsed right middle lobe with bronchiectasis and involvement of inferior bronchi of anterior segment of upper lobe

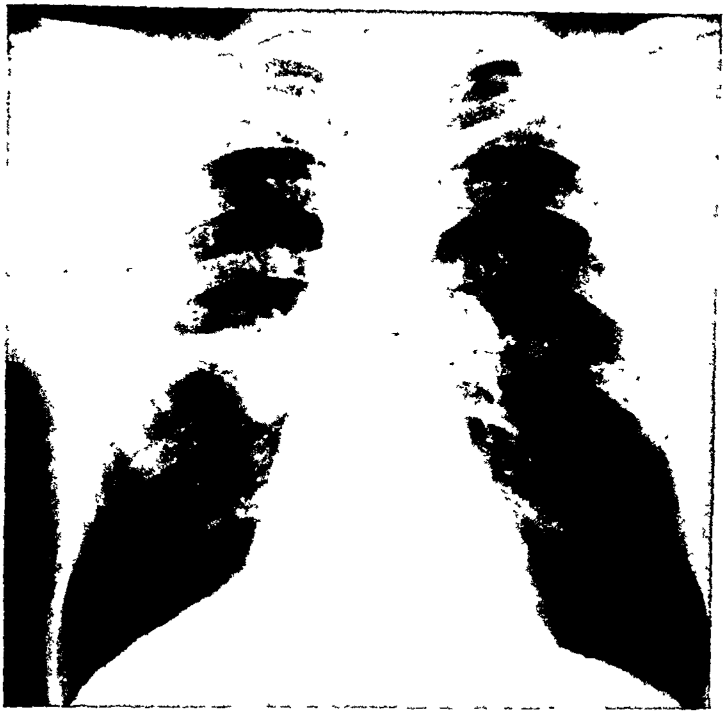


Fig. 1.—Roentgenogram of the chest which shows increased density and prominence of the lower pole of the right hilum, and some atelectasis of the right middle lobe.

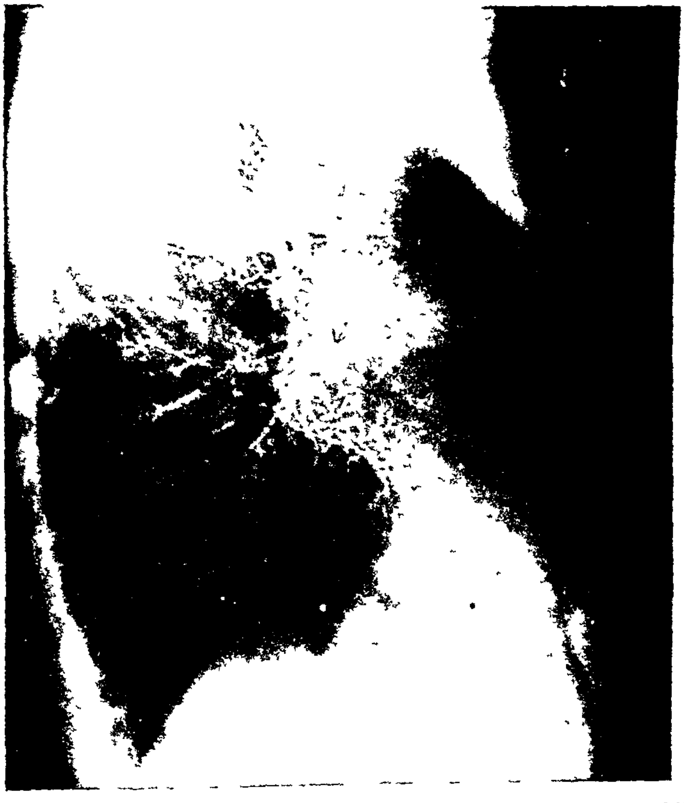


Fig. 2.—Right lateral bronchogram of chest which shows occluded right middle lobe bronchus and overexpansion of right lower lobe.

# A NOTE ON THE PROGNOSIS OF PULMONARY AGENESIS AND HYPOPLASIA ACCORDING TO THE SIDE AFFECTED

ALEXANDER J. SCHAFFER, M.D., AND ROWLAND V. RIDER, M.D., D.P.H.
BALTIMORE, MD.

A PRELIMINARY review of 30 reported cases of pulmonary agenesis largely from the pediatric literature, suggested to one of us that congenital absence of the left lung produced fewer symptoms than did absence of the right lung and that death occurred earlier and with greater frequency when the right side was affected (Table I).

TABLE I. 30 CASES OF PULMONARY AGENESIS

| LUNG ABSENT | NO. | SYMPTOMATIC | ASYMPTOMATIC | DIED | SURVIVED |
|---|---|---|---|---|---|
| Left | 17 | 11 | 6 | 2 | 15 |
| Right | 13 | 11 | 2 | 8 | 5 |

From such a small series no firm conclusions were drawn but the figures appeared to be highly suggestive. Suggestive also were comments frequently met in the literature concerning the greater frequency of left-sided over right-sided involvement. Both DeWeese and Howard[1] and Field[2] state that the majority of cases involve the left lung. Valle and Graham,[3] after reviewing 38 cases, stated that agenesis was encountered twice as often involving the left side as the right. One wondered whether this numerical difference had been present at birth or whether it appeared later in life because of difference in mortality between the two groups.

The publication in 1953 of Oyamada, Gasul, and Holinger,[4] in which the authors summarized all of the published data, historical, clinical, and pathologic on all the cases reported to date, presented an easy opportunity for exploring this question further. Their finding that 109 cases were divided almost equally into 50 with right-sided and 59 with left-sided involvement indicates that the over-all incidence of the disorder is not significantly greater on one side than the other.

Oyamada and his associates presented for each case the source of their information, a brief description of the findings including the side affected, and the age at which death occurred or the age when the patient was last known to be alive. Reported cases of doubtful diagnosis were listed separately and are not included in the analysis below. Those cases considered well established they presented in three groups: Group 1, cases of aplasia on one lung, Classes One and Two (Schneider[5]); Group 2, cases of congenital hypoplasia of one or both lungs; and Group 3, cases of congenital absence of both lungs.

---

From the Department of Pediatrics, The Johns Hopkins Medical Scho
ment of Biostatistics, The Johns Hopkins School of Hygiene, Baltimore, I

Received for publication March 13, 1956.

Interval postoperative examinations indicated that the *patient's condition has remained* good, his symptoms are minimal, and roentgenograms of the chest are satisfactory. In December, 1955, over ten years postoperatively, the patient wrote that he was feeling fine and working normally.

## SUMMARY

It is important to recognize benign bronchial tumors and treat them promptly. Although histologically benign, these tumors may cause death from bronchial obstruction which results in atelectasis, bronchiectasis, pneumonitis, suppuration, and possible hemoptysis. Lipoma is one of the rarer benign bronchial neoplasms, the case reported being the fourteenth on record. The tumor should be excised, either bronchoscopically or transthoracically. Persistent significant bronchiectasis may require pulmonary excision despite bronchoscopic removal of the tumor. This was necessary in our case, following which the patient has remained well for ten years.

## REFERENCES

1. Di Rienzo, S.: Radiologic Exploration of the Bronchus, Springfield, 1949, Charles C Thomas, Publisher, pp. 55-80.
2. Feller, A.: Über ein lipomähnliches hämartom der lunge, Virchow's Arch. f. path. anat. 236: 470, 1922.
3. Hochberg, L. A. and Schacter, B.: Benign Tumors of the Bronchus and Lung, Am. J. Surg. 89: 425, 1955.
4. Honig, A.: Cited by Vinson and Pembleton.[15]
5. Jackson, C. and Jackson, C. L.: Benign Tumors of the Trachea and Bronchi, With Especial Reference to Tumor-like Formations of Inflammatory Origin, J. A. M. A. 99: 1747, 1932.
6. Kernan, J. D.: Three Unusual Endoscopic Cases, Laryngoscope 37: 62, 1927.
7. Kirklin, J. W., McDonald, J. R., Clagett, O. T., Moersch, H. J., and Gage, R. P.: Bronchogenic Carcinoma Cell Type and Other Factors Relating to Prognosis, Surg., Gynec. & Obst. 100: 429, 1955.
8. Laboulbene, J. J. A.: Cited by Touroff and Seley.[14]
9. McGlade, T. H.: Fibro-lipoma of the Bronchus. Report of a Case, Ann. Otol., Rhin. & Laryng. 48: 240, 1939.
10. Myerson, M. C.: *Benign Neoplasms of the Bronchus. Report of a Case of* Fibrolipoma of the Left Main Bronchus Removed Through the Bronchoscope, Am. J. M. Sc. 176: 720, 1928.
11. Ochsner, S. and Ochsner, A.: Bronchial Adenoma (to be published).
12. Rokitansky, C.: A Manual of Pathological Anatomy, London, 1854, Sydenham Society, vol. 1, pp. 195-197.
13. Shanks, S. C. and Kerley, P.: A Textbook of X-ray Diagnosis, ed. 2, Philadelphia, 1951, W. B. Saunders Company, vol. 2, pp. 602-606.
14. Touroff, A. S. W. and Seley, G. P.: Lipoma of the Bronchus and the Lung. A Report of Two Unusual Cases, Ann. Surg. 134: 244, 1951.
15. Vinson, P. P. and Pembleton, W. E.: Lipoma of Left Main Bronchus. Report of a Case and Review of Literature, Arch. Otolaryng. 35: 868, 1942.
16. Watts, C. F., Clagett, O. T., and McDonald, J. R.: Lipoma of the Bronchus: Discussion of Benign Neoplasms and Report of Case of Endobronchial Lipoma, J. THORACIC SURG. 15: 132, 1946.
17. Wessler, H. and Rabin, C. B.: Benign Tumors of the Bronchus, Am. J. M. Sc. 183: 164, 1932.
18. Whalen, E. J.: Lipoma of the Bronchus, Ann. Otol., Rhin. & Laryng. 56: 811, 1947.

Their Group 3 furnishes no data for the question at hand. Data from their first two groups, therefore, form the basis of the following analysis. Seven fetal deaths were excluded from their total (4 patients with the left side affected and 3 with the right). The cases of bilateral hypoplasia have been classified according to the side most severely affected. The analysis was carried through for Groups 1 and 2 separately, but since the results are essentially the same, they are combined here.

Table II summarizes the evidence from these data and shows (compare Columns 4 and 7) that the prognosis seems considerably more favorable for left-sided agenesis. Column 8 indicates that by the age of 10 the difference has become statistically significant at about the 5 per cent level or less.

The conclusions are stated tentatively rather than definitively because of the possibility of unknown bias. For example, is there a larger fraction of the cases of agenesis of one lung that is asymptomatic and therefore does not come to autopsy or does not receive careful study and diagnosis? If the answer is the one indicated by the preliminary survey, that left-sided agenesis is more apt to be asymptomatic, and if a lower risk of dying is associated with lack of symptoms, the differences we found would be even more favorable to absence of the left lung. One should note that general underreporting of cases, with left and right side equally underreported, could change the apparent level of risk as well as the size of the ratio of left-sided to right-sided risk, though it would not change the direction of the difference.

The differential risk cannot be attributed to a greater incidence of other abnormalities among the patients with the right side affected, on the basis of the case descriptions in the Oyamada report.

It appears justified, therefore, to expect a greater survival rate when the left rather than the right lung is absent. The explanation for this difference might lie in the fact that the heart and mediastinal contents must move a greater distance to arrive at a position abutting the right axillary wall than the left. This greater magnitude of shift must carry with it greater distortion in the course of the great vessels and of the trachea and major bronchi, and a stronger possibility of kinking and perhaps vascular compression of the airway. This latter factor appears to be the one of greatest importance since most of the morbidity and mortality center about respiratory infection.

Following this line of reasoning it could be argued that mortality, especially in instances of right-sided agenesis, might be reduced by preventing the mediastinal contents from swinging too far from the midline. Perhaps this could be accomplished by the introduction of some nonirritating, nonabsorbable material, such as Lucite balls, into the affected hemithorax.

## SUMMARY

Statistical analysis of cases of pulmonary agenesis reported indicates that agenesis of the right lung carries a worse prognosis than agenesis of the left lung. It is suggested that this may be due to greater shift of heart and mediastinal contents with greater distortion of the course of blood vessels, trachea, and bronchi.

TABLE II. SURVIVAL OF CASES OF AGENESIS OF THE LUNG ACCORDING TO SIDE AFFECTED (102 Cases)

| AGE OBSERVED | LEFT SIDE AFFECTED (54 CASES) | | | RIGHT SIDE AFFECTED (48 CASES) | | | COMPARISON OF COLUMNS 4 AND 7 $\frac{\text{(DIFFERENCE)}}{\text{S.E. OF DIFF.}}$ |
|---|---|---|---|---|---|---|---|
| | NUMBER LAST OBSERVED AND ALIVE IN INTERVAL BETWEEN THIS AGE AND NEXT STATED AGE | NUMBER DYING IN INTERVAL BETWEEN THIS AGE AND NEXT STATED AGE | PERCENTAGE OF PERSONS DIAGNOSED WHO SURVIVED TO THIS AGE | NUMBER LAST OBSERVED AND ALIVE IN INTERVAL BETWEEN THIS AGE AND NEXT STATED AGE | NUMBER DYING IN INTERVAL BETWEEN THIS AGE AND NEXT STATED AGE | PERCENTAGE OF PERSONS DIAGNOSED WHO SURVIVED TO THIS AGE | |
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| 0 | — | 10 | 100.00 | — | 11 | 100.00 | — |
| 4 weeks | — | 3 | 81.48 | — | 6 | 77.08 | 0.54 |
| 3 months | 1 | 7 | 75.92 | 5 | 9 | 64.58 | 1.26 |
| 1 year | 8 | 2 | 62.80 | 3 | 5 | 44.19 | 1.90 |
| 10 years | 3 | 3 | 58.47 | 2 | — | 29.93 | 2.88 |
| 20 years | 2 | 5 | 50.31 | 2 | 4 | 29.93 | 1.98 |
| 40 years | 2 | 8 | 34.59 | 1 | — | 9.98 | 2.49 |
| Total cases | 16 | 38 | | 13 | 35 | | |

The data are from Tables I and II of ref. 1; for method of calculating Columns 4 and 7 and the standard errors of the percentages in these columns, see ref. 6.

# THE RESULTS OF PULMONARY RESECTION IN THE TREATMENT OF TUBERCULOSIS: AN EVALUATION OF 201 CONSECUTIVE RESECTIONS*

JACK C. COOLEY, M.D.,** FRITZ H. MOSER, M.D.,*** ROCHESTER, MINN., AND
GUSTAF A. HEDBERG, M.D.,† NOPEMING, MINN.

THE past decade has seen many pertinent changes in the management of the patient with pulmonary tuberculosis.[1-3] Salient among these has been the advancement and application of thoracic surgery as an important adjunct to the medical and sanatorium regimen. Equally important has been the development and judicious use of the chemotherapeutic drugs, both in the general management of the tuberculous patient and in his preoperative and postoperative phase.[4] Certainly, the chemotherapeutic drugs have improved the results of excisional surgery in tuberculosis and at the same time have reduced both mortality and morbidity. Many patients previously ineligible for excisional therapy because of widespread, bilateral, or endobronchial disease have been improved by these drugs to the extent that resection has become feasible.

With the foregoing considerations in mind, it seemed timely to review the cases in which pulmonary resection has been used at the Nopeming Sanatorium in conjunction with chemotherapy in the treatment of pulmonary tuberculosis. The years 1951 through 1954 were chosen for this study because this period of four years represents a time when the combination of surgery and chemotherapy was used extensively in the management of the tuberculous patient. All cases in which operation was performed during this time are reported.

Indications for surgical therapy were gradually broadened during the 4-year period over which this study was made. All decisions to use surgical treatment for any given patient were made by the conference-selection method, in which all staff members of the sanatorium participated. The great majority of indications for resective surgery involved the fibrocaseous focus or cavitary lesion remaining following an active sanatorium and chemotherapeutic regimen of a varied duration. Tuberculous endobronchial disease and the residue thereof in the form of tuberculous bronchiectasis and bronchostenosis were the indications for surgical treatment in a few cases.

The preoperative duration of chemotherapy varied from several weeks to several years. A further breakdown of the total duration of chemotherapy in these cases will be included later.

---

The Mayo Foundation, Rochester, Minn., is part of the Graduate School of the University of Minnesota.

Received for publication March 19, 1956.

*Pulmonary resections were carried out by Drs. Josiah Fuller, N. K. Jensen, W. R. Schmidt, J. W. Kirklin, and T. J. Kinsella.

**Fellow in Surgery, Mayo Foundation, Rochester, Minn.

***Fellow in Surgery, Mayo Foundation, Rochester, Minn.

†Deceased. Formerly at Nopeming Sanatorium, Nopeming, Minn.

## REFERENCES

1. DeWeese, E. R., and Howard, J. C.: Congenital Absence of a Lung Diagnosed Before Death, Radiology 42: 389, 1944.
2. Field, C. E.: Pulmonary Agenesis and Hypoplasia, Arch. Dis. Childhood 21: 61, 1945.
3. Valle, A. R., and Graham, E. A.: Agenesis of the Lung, J. THORACIC SURG. 13: 345, 1944.
4. Oyamada, A., Gasul, B. M., and Holinger, P. H.: Agenesis of the Lung, Am. J. Dis. Child. 85: 182, 1953.
5. Schneider, P., in E. Schwalbe: Die Morphologie der Missbildungen des Menschen und der Tiere, Jena, 1909-1913, Gustav Fischer, vol. 3, part 2, p. 772.
6. Merrell, M. and Shulman, L. E.: Determination of Prognosis in Chronic Disease, J. Chron. Dis. 1: 12, 1955.

obstructive pulmonary complications. Bronchoscopy was reserved for those cases in which definite roentgenologic and clinical signs of retention of secretion and atelectasis developed.

*Mention should be made that tracheotomy has been found useful in the* postoperative management of some of these problems. Particularly is this true in the older patient with some emphysema, retention of secretion, and air leak with or without subcutaneous emphysema. Although tracheotomy was used in only 2 patients in this series, subsequent experience has proved its value.

### CLINICAL FEATURES

The files of the Nopeming Sanatorium contained data on 189 patients who underwent pulmonary resection from January, 1951, through December, 1954, as part of the regimen planned in the treatment of their pulmonary tuberculosis. Twelve of these patients underwent bilateral pulmonary resection nonsimultaneously, bringing the total number of resections to 201. The ages of these patients varied from 8 to 65 years, with the heavy distribution occurring in the third, fourth, and fifth decades. Table I includes the exact age distribution. The sex ratio was almost equal, there being 92 male and 97 female patients.

Classification was made of each patient according to whether the disease was minimal, moderately advanced, or far-advanced tuberculosis. The severity of the disease submitted to resection is somewhat indicated by the fact that 59 patients were classified as having far-advanced disease, 93 as having moderately advanced disease, and only 37 with minimal disease. Bacterial proof of tuberculosis was of course attempted preoperatively in all cases by sputum and gastric studies. In 181 patients, *Mycobacterium tuberculosis* was isolated, while in eight cases no bacterial proof of tuberculosis was found. Operation was performed in these eight cases because clinically, roentgenographically, and by response to treatment, the patients were all thought to have tuberculosis. *All 8 patients were found to have fibrocaseous lesions pathologically and* 4 had cavitation. Seven of these 8 had positive bacteriologic findings on the resected specimen, confirming our roentgenographic and clinical impressions.

TABLE I. AGE RANGE OF 189 PATIENTS UNDERGOING PULMONARY RESECTION

| AGE (YEARS) | PATIENTS |
|---|---|
| 0-9 | 1 |
| 10-19 | 14 |
| 20-29 | 42 |
| 30-39 | 61 |
| 40-49 | 43 |
| 50-59 | 26 |
| 60-69 | 2 |

Chemotherapy, as previously indicated, consisted primarily of combinations of three drugs: streptomycin (or dihydrostreptomycin), para-aminosalicylic acid, and isoniazid. Each patient in this series received some chemotherapy, either preoperatively or postoperatively or, more commonly, both. The patients who underwent operation in the past two years have received

The main chemotherapeutic drugs employed in the treatment of this surgical series included streptomycin (or its derivative dihydrostreptomycin), isoniazid, and para-aminosalicylic acid. Generally speaking, two of these drugs were used at any given time unless contraindicated by untoward reactions. In a few cases, all three drugs were employed simultaneously. Several other antituberculous drugs were used when specific sensitivity or resistance studies indicated their possible efficacy. These occasionally used drugs included viomycin, tibione, and more recently pyrazinamide.

All patients undergoing surgical therapy were submitted to bronchoscopy in the preoperative period. Pulmonary function and bronchospirometric studies were used preoperatively in those patients whose respiratory reserve was borderline or indefinite. Inadequate respiratory reserve, as determined by these tests and clinical examination, was a contraindication to thoracic surgical therapy. Bronchography was carried out whenever indicated, using chloriodized oil (Iodochlorol) or Dionosil as the opaque medium. An attempt was made in all cases of tuberculous bronchiectasis to map out accurately all segments of both lungs in order to evaluate better the indications for, and the extent of, pulmonary resection.

The surgical procedure was carried out with the patient in the prone position using the Naclerio headrest. In almost all cases a rib was resected. Major bronchi, including segmental bronchi, were closed by use of interrupted through-and-through sutures of silk in an end-on closure after the method of Sweet.[5] Subsegmental resection was utilized, when efficacious, by isolating and ligating the subsegmental bronchovascular bundle and then applying curved clamps to the subsegmental boundaries and excising the tissue thus isolated. Closure of the lung was then effected by a two-row continuous catgut suture, the clamps being used as a guide for the first row. Care was taken to close, by silk-suture ligatures, all known air leaks following resection. Careful expansion of all remaining segments of the lung on the side of operation was required before the chest was closed. Three catheters with extra fenestrations at their ends were inserted several interspaces below the line of incision through stab wounds. Two of these catheters were placed in the apical portion of the chest, one anteriorly and one posteriorly. The third catheter was placed for dependent drainage on the diaphragmatic surface. The catheters were attached immediately to a water-seal drainage system and later attached to gentle continuous suction after the patient was transported to his room. Usually no drainage was utilized in cases wherein pneumonectomy was carried out.

Postoperatively, suction at a pressure of 15 cm. of water was maintained on all chest catheters until they were removed. This was usually accomplished between the third and the fifth day, but of course the time of removal depended on the status of expansion of the lung and amount of fluid and air leakage present. Antituberculous chemotherapy was started as soon as feasible postoperatively. Penicillin was almost routinely used prophylactically, but broad spectrum antibiotic drugs were withheld unless specifically indicated. Forced coughing, steam, and tracheal aspirations were used as indicated to prevent

Previous surgical therapy for tuberculosis had been carried out in 27 patients prior to their resection. This consisted of thoracoplasty in all instances. In no other patients in this series had a resection, decortication, or other thoracic surgical procedure been done elsewhere prior to resection at the Nopeming Sanatorium. Thirty-eight thoracic surgical procedures were done as an adjunct to pulmonary resection. In 19 cases thoracoplasty was done. Seventeen of these were done at or just before the time of resection, while two were done after the patient's recovery from resection.

The incidence of postoperative complications was not high. Retention of secretion severe enough to require bronchoscopy was the commonest complication, occurring in eighteen cases (9 per cent). Persistent bronchopleural fistula occurred after eight resections (4 per cent). In four of these instances, surgical therapy of this fistula and thoracoplasty were required, while in the other four, thoracoplasty alone was utilized in the treatment. In none of the eight cases was the bronchopleural fistula felt to be from the larger bronchi. In all cases small bronchial leaks from the raw surface of the lung were thought responsible. Frank empyema occurred in 4 patients (2 per cent). Persistent air "pocket" and pneumothorax following removal of the chest tubes were two other less common complications. Table IV includes a classification of all complications occurring in this series.

TABLE IV. COMPLICATIONS OCCURRING IN 201 PULMONARY RESECTIONS

| COMPLICATION | PATIENTS |
|---|---|
| Retention of secretion requiring bronchoscopy | 18 |
| Bronchopleural fistula | 8 |
| Persistent "pneumo" pocket | 13 |
| Empyema | 4 |
| Pleural effusion | 3 |
| Hemorrhage with hemothorax | 3 |
| Wound infection | 3 |
| Pneumothorax | 6 |
| Transfusion reaction | 1 |
| Subcutaneous emphysema | 1 |
| Wound hematoma | 1 |
| Paralytic ileus | 2 |
| Psychosis | 1 |
| Guillain-Barré syndrome | 1 |

Twenty-five thoracic surgical procedures were necessary in the treatment of complications. These included thirteen thoracoplasties, five decortications, four closures of bronchopleural fistula combined with thoracoplasty, two tracheotomies, and one excision of a long bronchial stump.

### FOLLOW-UP RESULTS

All 189 patients were followed for periods ranging from two months to four and one-half years. Follow-up was accomplished by roentgenologic, gastric and sputum studies in the postoperative sanatorium phase, and then by gastric and roentgenologic studies at intervals of three to six months for varying periods following the patient's dismissal from the sanatorium. In eighteen almost routinely a minimum of four to six months of preoperative chemotherapy and an equal or greater duration, postoperatively. The average doses of the chemotherapeutic drugs included 1 Gm. of streptomycin or dihydrostreptomycin twice a week, 100 mg. of isoniazid three times daily, and 12 to 14 Gm. of sodium para-aminosalicylic acid, daily, in divided doses. Table II indicates a breakdown by duration of time of the total amount of chemotherapy administered.

TABLE II. TOTAL DURATION OF CHEMOTHERAPY

| MONTHS OF CHEMOTHERAPY | PATIENTS |
|---|---|
| 0-6 | 16 |
| 6-12 | 58 |
| 12-18 | 64 |
| 18 or more | 51 |

## SURGICAL FEATURES

Two hundred and one thoracotomies were carried out in this series. In each case resection of some lung tissue was done. A classification of the various types of pulmonary resection would include subsegments, segments, lobes, and pneumonectomy. According to the indications, combinations of these resections were carried out in the individual case at operation. Table III, although not exacting in its breakdown of the types of resection used in any individual case, does give some indication of the over-all amount of pulmonary tissue removed. Pathologic reports were available on all but 3 patients. Many specimens contained more than one type of tuberculous lesion. Fibrocaseous-nodular and cavitary disease were the two types reported most frequently, while a smaller number of specimens showed bronchiectasis, active tuberculous bronchitis, bronchostenosis, or obstructive pneumonitis. Interestingly enough, 2 patients were found to have concomitant bronchogenic carcinoma with their tuberculosis. Both were treated surgically as if the lesion had been carcinoma only.

TABLE III. AMOUNT OF LUNG RESECTED IN 189 PATIENTS UNDERGOING PULMONARY RESECTION

| TYPE OF RESECTION | RIGHT | LEFT | TOTAL |
|---|---|---|---|
| Subsegments | 56 | 45 | 101 |
| Segments | 75 | 78 | 153 |
| Lobes | 28 | 16 | 44 |
| Pneumonectomy | 7 | 6 | 13 |

Bacteriologic studies on all resected specimens were carried out. Such studies include smears, cultures for *Mycobacterium tuberculosis,* and guinea pig inoculations. Reports on these studies were available on all but 22 patients. Forty-seven patients had a positive smear only, while 32 had the guinea pig inoculation or culture positive only. Both smear and cultural methods were positive in 43. All attempts to isolate the tuberculosis bacillus from the specimen were negative in 57.

negative bacteriologically while in the sanatorium postoperatively, but subsequently were positive on sputum or gastric studies and were readmitted for further treatment

A point to be emphasized is that of twelve bilateral pulmonary resections carried out in this series, three were in the failure group This represents a failure rate of 25 per cent when bilateral resection was used in the therapy of tuberculosis and indicates that great care must be taken in evaluating patients for bilateral resective therapy

## COMMENT

The over all results of pulmonary resection for tuberculosis are good A recent review of the results in 189 patients with tuberculosis treated by a standard sanatorium regimen, chemotherapy, and pulmonary resection and herein presented has helped solidify our opinion in this regard Certainly only a part of the credit can be allocated to resective surgery Good sanatorium care and a well planned chemotherapy program are an integral part of the over all management of the tuberculous patient undergoing pulmonary resection

## REFERENCES

1 Conklin, W S Surgical Trends in Pulmonary Tuberculosis, Dis Chest 27 147 164, 1955
2 Decker, A M, Raleigh, J W, and Welles, E S The Coordination of Surgery and Combined Chemotherapy in the Treatment of Pulmonary Tuberculosis, J THORACIC SURG 29 151 158, 1955
3 Hedberg, G A Changing Concepts in the Treatment of Tuberculosis, M Clin North America July, 1954, pp 1161 1166
4 Douglass, R, Bosworth, E B, Judd, J M, and Chang, K H Resection Surgery in Tuberculosis Complications and After History, J THORACIC SURG 29 136 150, 1955
5 Sweet, R H Thoracic Surgery, Philadelphia, 1954, W B Saunders Company, 368 pp

instances, resection was carried out on patients who were transferred to Nopeming for surgical treatment from other nearby sanatoriums. In all of these cases, follow-up was by contacting the respective sanatorium to which the patient had returned. In seven cases, follow-up was roentgenologic only, because for one reason or another gastric studies were declined. There were eight deaths in all, representing a 4.2 per cent over-all mortality. Of these, 5 patients (2.6 per cent) died in the sanatorium, while 3 died at various time intervals following their dismissal from the sanatorium. The causes of the postoperative early deaths include one case each of cardiovascular accident, severe subcutaneous emphysema, cardiac arrest which did not respond to cardiac massage, postoperative vascular collapse, and Guillain-Barré syndrome. The cause of two of the late deaths was myocardial failure in one patient and an automobile accident in another. The cause of the third late death was indefinite, and unfortunately no post-mortem examination was made. The death was not thought to be due to tuberculosis, however.

Good results, with continued sputum conversion and roentgenologic stability of residual lesions, were found in 160 of the 189 patients (85 per cent). An additional 7 patients were followed roentgenologically only, and all of these appeared to have a good result as determined by the roentgenologic and clinical stability. The total number of patients with good results is thus 167 (88 per cent) if these two groups are considered together. A more accurate appraisal of these good results is gained from Table V, in which they are classified according to the duration of follow-up since resection.

TABLE V. PATIENTS CLASSIFIED AS HAVING A "GOOD RESULT" FOLLOWING PULMONARY RESECTION

| TIME FOLLOWED | GASTRIC STUDIES | ROENTGENOLOGIC STUDIES ONLY |
|---|---|---|
| 0-6 months | 19 | 0 |
| 6-12 months | 74 | 2 |
| 1-2 years | 57 | 2 |
| 2-3 years | 9 | 0 |
| 3-4 years | 1 | 2 |
| 4-5 years | 0 | 1 |

Thirteen patients (6.9 per cent) had recurrent bacterial positivity following resection, and the results for these patients were classified as unsatisfactory. One additional patient had negative sputum and gastric studies, but had a change in the chest roentgenogram which strongly suggested activity in the residual pulmonary lesion. This patient has also been classified as having an unsatisfactory result. It was of interest to us that, of the fourteen "resection failures," 8 remained positive postoperatively in the sanatorium. Two of these have subsequently been found bacteriologically negative following further chemotherapy and dismissal from the sanatorium. A critical review of this group of 8 patients suggests that perhaps they represent more a failure in patient selection than a failure of pulmonary resection. Five patients were the complication of a residual space, the surgical procedures were segmentectomy in 22, subsegmental resections in 6, and wedge resections in 6. Decortication was performed as an associated procedure in 5 of these cases. Thirty of the complications were in male patients and 3 in females, and the average age of the group was similar to the series as a whole.

Persistent cavitary or nodular disease represented the primary indications for resection in the entire group of patients. It should be noted, however, that in those patients with complications, there was a higher incidence of decortication necessitated by previous pneumothorax or effusion than in the entire group. All of the patients had received antibiotics and chemotherapy prior to resection for 1 to 18 months, with an average of 9 months preoperative therapy. Various combinations of drugs were about equally represented in this group of patients and they were treated from 3 to 18 months postoperatively, with an average of 4½ months therapy after surgery.

The majority of the residual spaces were noted to be present within 72 hours, however, at least one patient developed a space on each succeeding day up to the tenth postoperative day. All of these spaces developed in spite of the routine use of multiple chest catheters for subaqueous negative pressure drainage of the pleura. Once the space was recognized, negative pressure drainage was continued if present until it was considered to be ineffective, when the catheters were removed and the space was then studied by roentgen methods, needle aspiration, the injection of foreign material to determine the presence of fistula and/or reinsertion of catheters in the space.

In 34 persistent spaces, the original chest tubes were considered effective for from 3 to 21 days postoperatively, with an average of 7 days. Multiple thoracenteses were considered effective in treating the complication in fourteen instances. Chest catheters were reintroduced into the space for continued closed drainage sixteen times for a period of 1 to 17 days with an average of 9 days' duration. There were 4 patients who had no further therapy.

Specific evidence of bronchopleural communication was demonstrated in 8 of the patients and, of this group, 2 had open drainage performed when evidence of infection was found. Of the 2 latter patients, one had a small thoracoplasty performed. None of the other patients had further surgical procedures.

The technique of handling the intersegmental plane was equally represented in the patients with persistent spaces. About one third of the patients had careful closure of the intersegmental or bare area by suturing the edges of the wound. In another 9 cases the bare area or intersegmental plane was not treated except for particular hemostasis and closure of small leaks on the surface; the remaining patients had the bare surface covered by an attached flap of pleura.

The length of time that the pleural pocket was seen roentgenographically varied from 10 to 937 days, with an average presence of 97 days. The duration of the space in the 8 patients in whom a bronchopleural fistula large enough to be demonstrated by objective means varied from 23 to 269 days, with an average of 169 days.

# THE FATE OF THE RESIDUAL PLEURAL SPACE FOLLOWING SMALL RESECTIONS FOR PULMONARY TUBERCULOSIS

Wilson Weisel, M.D., Robert F. Thimmig, M.D., and
Raymond R. Watson, M.D.
Milwaukee, Wis.

The presence of a residual pleural space due to incomplete lung re-expansion following lung resections for pulmonary tuberculosis has been considered a potentially serious complication of the procedure.[1-3] The danger of its appearance lies in the presumed likelihood of bronchopleural fistula, empyema, overdistention of remaining lung and concomitant spread of tuberculous disease. Because of these dangers, various collapse procedures have been recommended to be performed concurrently with surgery or soon after the space has been detected.

In order to evaluate the status of these views, a policy of careful observation and follow-up was adopted in caring for postoperative patients with a residual pneumothorax or pleural space. The decision for further operative procedure was made dependent only on the development of an empyema or other superimposed complication of comparable magnitude.

The series of patients utilized for this study was 100 consecutive cases of small resections performed on 90 patients as primary major surgical procedures between November, 1950, and November, 1954. The age of the group ranged from 23 to 63, with an average of 43 years. The sex distribution was 72 males and 18 females. The only selection exercised consisted of excluding patients who had had previous major surgery, such as thoracoplasty, or patients in whom the resection involved one complete lobe or more.

Residual air pockets which were of sufficient size to be easily recognized on postoperative x-rays were noted following 34 resections in 28 patients. These were present or developed more than 72 hours after resection. All of these patients were carefully followed in the immediate postsurgical period and have been rechecked to the present time to determine the occurrence of late complications. The shortest period of postoperative examination has been 14 months and the longest 5½ years. We have considered all of these spaces to represent bronchopleural fistulas and, regardless of their appellation, we believe this is the best explanation of their pathogenesis. In addition, because of this pathology, we feel further that careful, regular, and persistent follow-up examinations including history, physical examination, thoracic roentgenograms, and gastric cultures are imperative.

The primary surgical procedures in the patients studied were segmental resection in 70, subsegmental in 18, and wedge resection in 12. Decortication was performed as a concomitant procedure 12 times. In the patients with

---

From the Surgical Service, Veterans Administration Hospital, Wood, Wis., St. Joseph's Hospital and Columbia Hospital, and the Department of Surgery, Marquette University School of Medicine.

Received for publication March 19, 1956.

Of the 28 patients who presented spaces, 2 were found to have positive gastric cultures 4 and 1½ years postoperatively, respectively It might be said, however, that both patients had rather extensive bilateral disease originally, no obvious active parenchymal lesion was apparent in current x rays and the tubercle bacilli cultured have not reproduced tuberculosis in the animal *Single episodes of hemoptysis have occurred in 2 patients* at intervals of 3 and 2 years after surgery, respectively No definite connection with the previous surgical complication has been established in these patients and no evidence of active pulmonary disease has been seen radiographically The late complications in this group of patients have seemed slightly higher than would be anticipated ordinarily but when the type of original disease was considered this was not unexpected

To demonstrate this group of cases the following case histories are cited

## CASE REPORTS

CASE 1—P H, 136424, a 61 year old white man, was admitted Jan 9, 1952, from another institution because of moderately advanced, active, bilateral pulmonary tuberculosis He demonstrated cavitary disease of the right upper lobe and positive sputum (Fig 1) He was treated for 12 months with streptomycin and para aminosalicylic acid (PAS) and on April 8, 1952, a right apical and posteror segmentectomy was performed The lung appeared well expanded after surgery but on April 11, 1952, a moderate sized apical pleural space appeared The original catheters became ineffective on the fifth postoperative day and were removed (Fig 2) A new catheter was reinserted into the space and was effective for 10 days and it was then removed Although the patient did very well clinically the apical space was visible radiologically for 217 days He was discharged from the hospital on April 17, 1953 The last follow up examination on Oct 12, 1955, showed him to be in good health, he had no complaints, his chest x rays appeared normal (Fig 3), and gastric cultures have been consistently negative

CASE 2—F W, 147964, a 35 year old white man was admitted July 25, 1953, from another institution because of minimal, active, left fibronodular and cavitary pulmonary tuberculosis (Fig 4) On Dec 10, 1953, after 7 months' therapy with streptomycin and PAS, a left apicoposterior segmentectomy was performed Although the lung was well expanded after operation, a pleural space was evident 2 days after surgery The chest catheters functioned for 4 days and were then removed (Fig 5) Thoracenteses were performed on 8 occasions with removal of moderate quantities of air and no fluid but a fistula could not be demonstrated by the injection of methylene blue, saline, or ether The space persisted for 104 days The patient was discharged asymptomatic and with negative sputum on Nov 5, 1954 Recent examinations on Aug 10 and Nov 5, 1955, have found the patient to be without symptoms, chest x rays (Fig 6) are stable, and gastric cultures are negative

CASE 3—J P, 141333, a 30 year old white man, was admitted to the hospital on Sept 4, 1952, because of moderately advanced, active, fibronodular pulmonary tuberculosis of the right lung (Fig 7) He had a previous history of severe alcoholism and recurring residences in correctional institutions On Oct 21, 1952, a subsegmental resection of the anterior segment of the right lobe was performed through a posterolateral thoracotomy for a filled cavity the contents of which were positive on culture for acid fast bacilli

Three days following surgery an apical pneumothorax space became apparent (Fig 8) No gross evidence of bronchial fistula could be definitely ascertained and no further treatment was performed The pleural space could not be seen on Dec 8, 1952, or 53 days after its appearance Follow up studies in July, 1955, presented no abnormal findings (Fig 9)

CASE 4—O B, 139480 This 31 year old white man was admitted to the hospital on June 4, 1952 He was transferred from another institution having been under treatment for

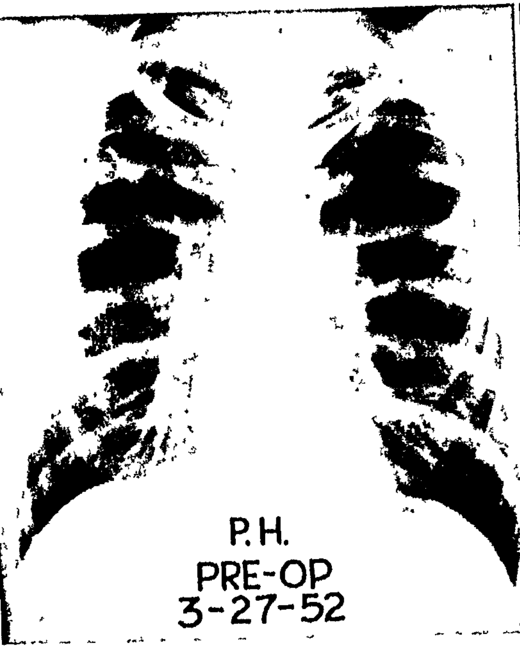


Fig. 1.

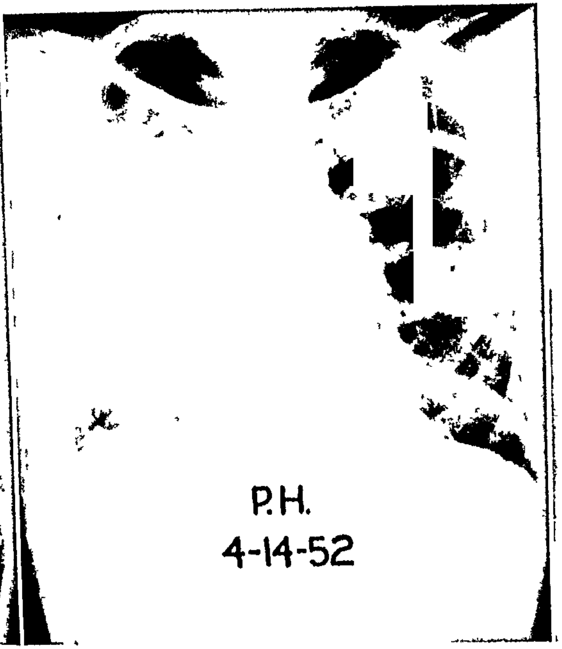


Fig. 2.

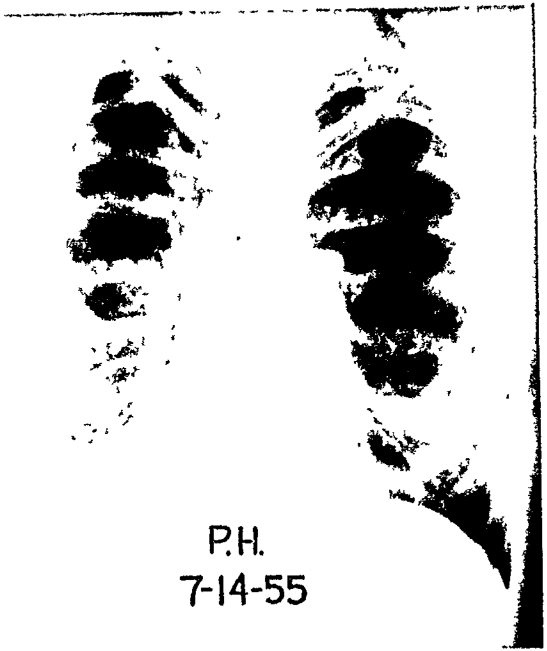


Fig. 3.

Fig. 1.—Posteroanterior thoracic roentgenogram showing residual cavitary disease of the right upper lobe following long-term chemotherapy.

Fig. 2.—Postoperative pneumothorax and pleural changes which occurred three days following right apical and posterior segmentectomy.

Fig. 3.—Chest x-ray showing stable appearance of right lung and resolution of pleural complications three and one-half years after segmentectomy.

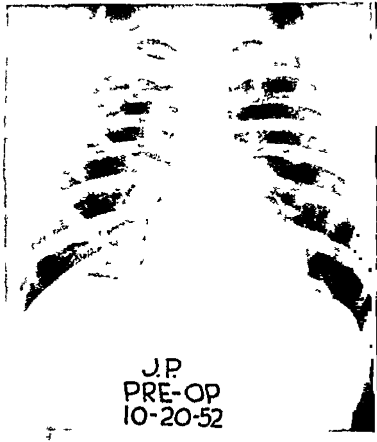


Fig 7

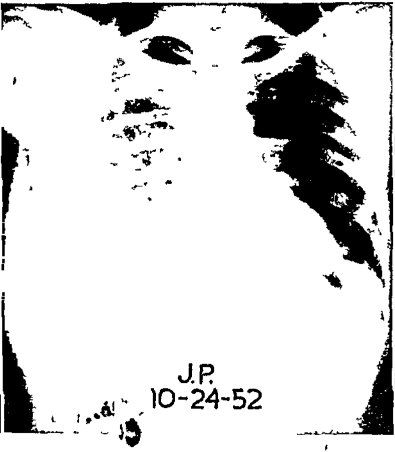


Fig 8

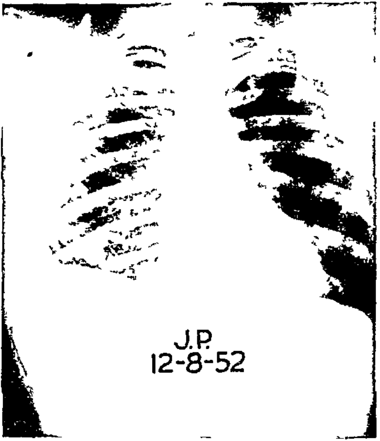


Fig 9

Fig 7—Posteroanterior chest x ray showing residual nodular disease in right second anterior interspace
Fig 8—Postoperative thoracic roentgenogram showing right apical pneumonthorax which appeared three days after operation
Fig 9—Chest x-ray three months after subsegmental resection of right upper lobe

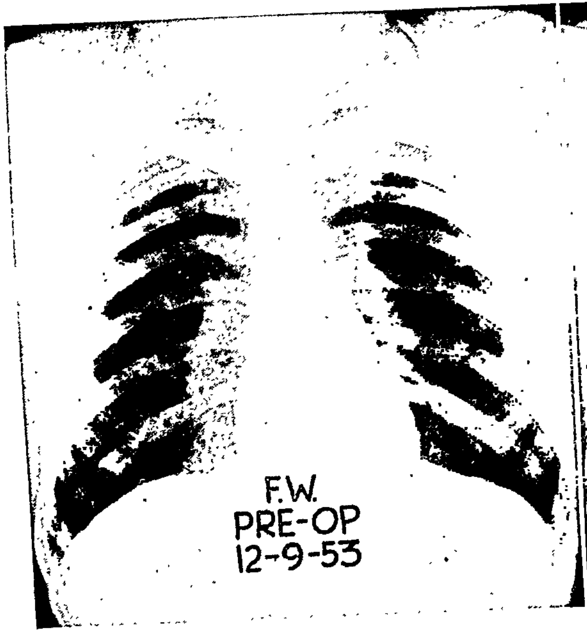


Fig. 4.

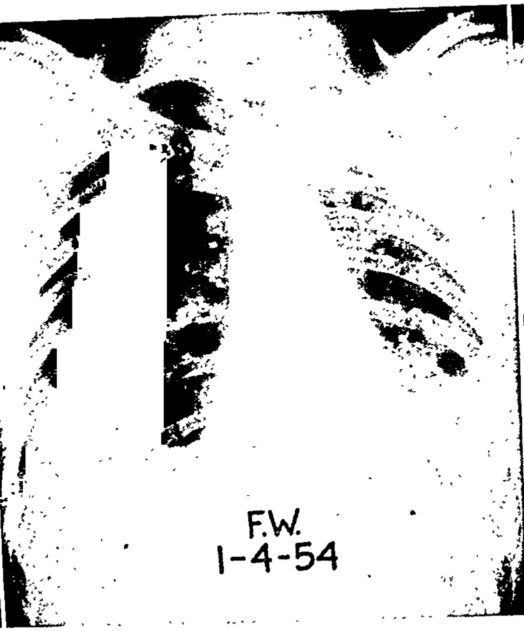


Fig. 5.

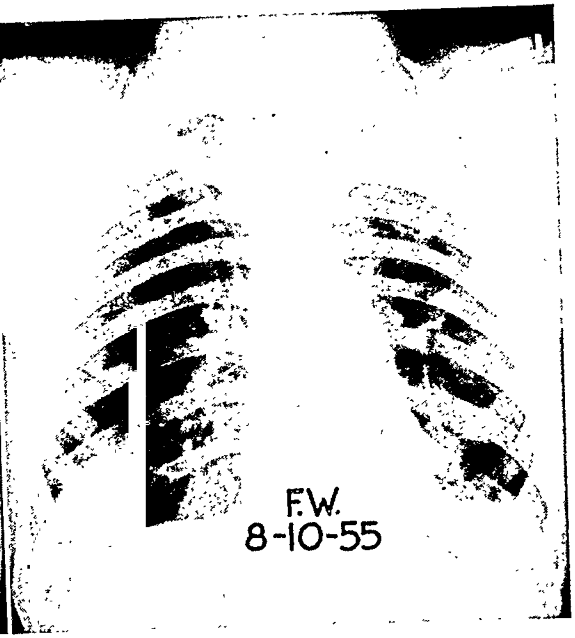


Fig. 6.

Fig. 4.—Residual fibronodular tuberculosis in left upper lobe following seven months' antibiotic and chemotherapy as seen in posteroanterior roentgenogram.

Fig. 5.—Apical pleural space which appeared ten days postoperatively as it was viewed twenty-five days following segmentectomy.

Fig. 6.—Stable appearance of left lung twenty months following segmentectomy showing change of pleural complications on chest x-ray.

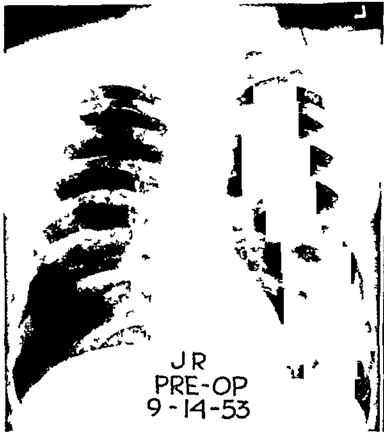


Fig 13

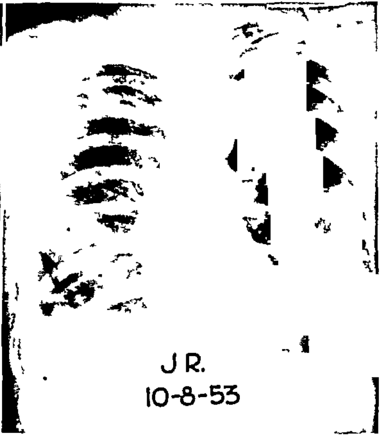


Fig 14

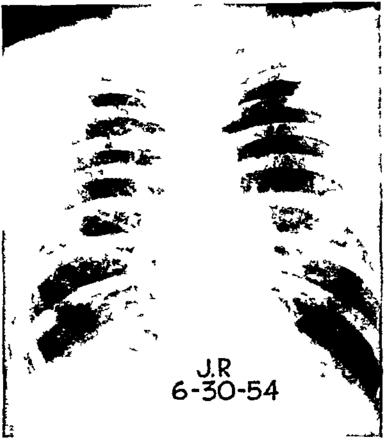


Fig 15

Fig 13—Fibrocavitary tuberculosis remaining in right upper lobe as seen on posteroanterior roentgenogram
Fig 14—Postoperative right apical space which appeared ten days following segmentectomy
Fig 15—Posteroanterior roentgenogram showing resolution of postoperative pleural complications

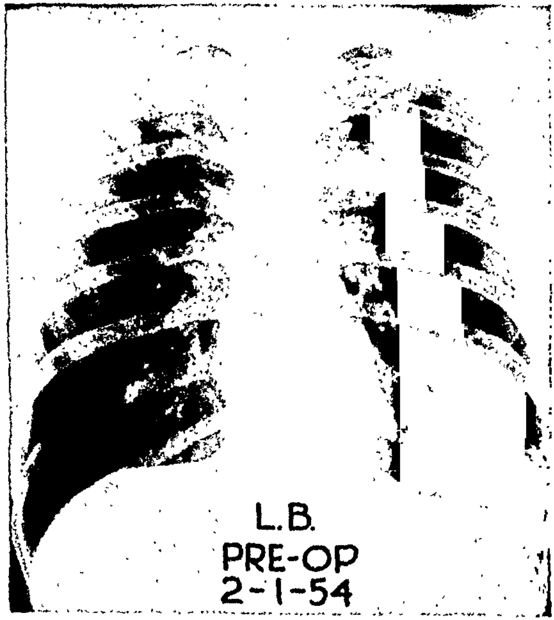


Fig. 10.

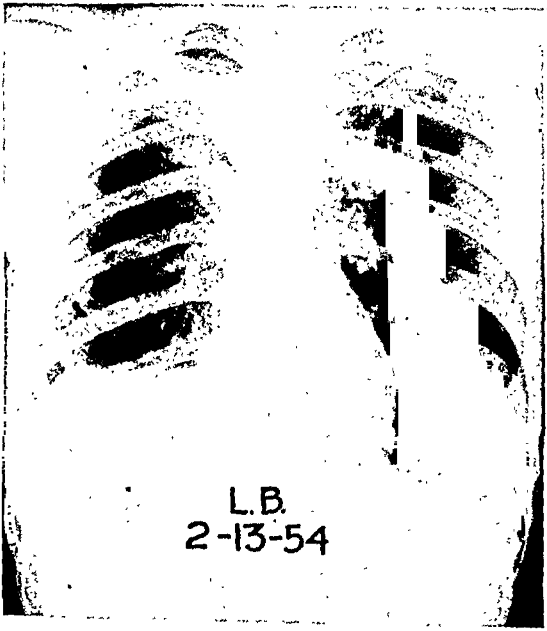


Fig. 11.

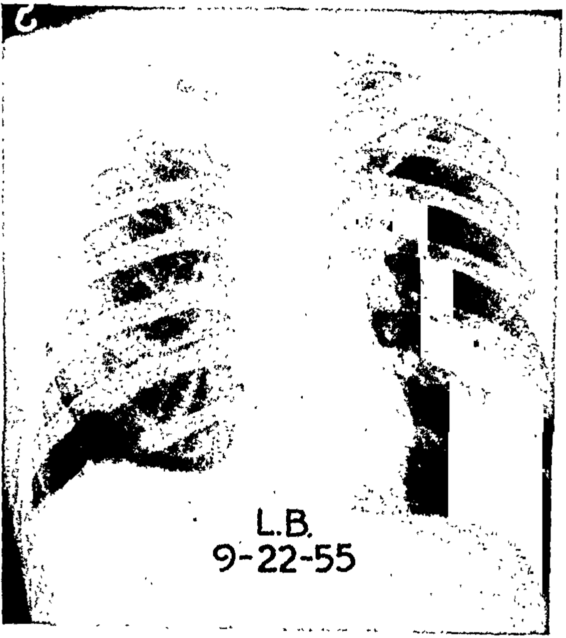


Fig. 12.

Fig. 10.—Posteroanterior thoracic roentgenogram showing persistent cavitary tuberculosis of the right upper lobe.
Fig. 11.—Apical pneumothorax on right, eleven days after right apical and posterior segmentectomy.
Fig. 12.—Postoperative chest x-ray showing clearing of postoperative pleural space on right.

# THE EFFECT OF PREOPERATIVE CHEMOTHERAPY ON THE INCIDENCE OF TUBERCULOUS LESIONS IN THE BRONCHI

WALLY GORDON, F.R.C.S. (EDIN.), F.R.C.S (ENG.)*
LONDON, ENGLAND

IN a previous communication, the author[1] showed that preoperative streptomycin reduces the incidence of bronchopleural fistula after pulmonary excision for tuberculosis and that the incidence of fistula is influenced by the duration of streptomycin treatment before operation. It was assumed that the reduced incidence of fistula was due to the effect of the preoperative streptomycin on the tuberculous lesions in the transected bronchi. However, at the time of that investigation, practical difficulties prevented a coincident study to determine the effect of chemotherapy on the histologic appearances of the excised bronchi. The aim of the present communication is to show that treatment with antibacterial agents does influence the incidence of tuberculous lesions in the bronchi.

Eighteen patients did not receive drugs before operation as they were treated before the period of routine chemotherapy. Fifty-one patients received such treatment before operation and 36 of these patients had received more than one course of drugs. In 32 patients, as the intervals between courses were long, only the last course was taken to represent the preoperative treatment. In 4 patients, as the interval between the penultimate and the final course was less than four weeks, the two courses, but excluding the interval between them, were considered together as the duration of preoperative drug treatment.

Three patients received streptomycin alone, 28 streptomycin and PAS (para-aminosalicylic acid), 14 streptomycin and INAH (isonicotinic acid hydrazide), 4 streptomycin, PAS and INAH, one INAH alone, and 2 patients received INAH and PAS. During part of the course, 6 patients were given 1 Gm. of streptomycin, thrice weekly. All other patients on streptomycin received 1 Gm. daily. Either 150 or 200 mg. of INAH and from 12 to 20 Gm. of PAS were given daily.

The 69 patients underwent pulmonary excision for tuberculosis and the central end of the divided bronchus was examined in each of the operation specimens (nineteen lungs, thirty-five lobes, and fifteen segments). The histologic examinations were routine and the patients providing the pathologic material were unselected.

In Table I the patients are grouped according to whether or not they received preoperative chemotherapy. In the group which did not receive drugs

---

Received for publication March 19, 1956

*Present address: The Lady Templer Chest Hospital, Kuala Lumpur, Malaya moderately advanced, bilateral, active, reinfective pulmonary tuberculosis with streptomycin and PAS for 3 months.

A left thoracotomy was performed on Aug. 26, 1952, to remove residual nodular disease in the upper and mid-lung fields. Subsegmental resections of the apicoposterior and superior segments were used to remove four fibrocaseous lesions. The postoperative course was satisfactory until the fifth postoperative day when a large apical pneumothorax space appeared. The latter was treated by insertion of an apical catheter and negative pressure suction for 2 weeks, but lung re-expansion did not occur although no definite evidence of a large fistula could be obtained. The pneumothorax space was seen radiologically until Oct. 24, 1952. A month later a right thoracotomy was performed with resection of three granulomatous lesions by wedge resection. The postoperative course was uneventful and the patient was prepared through the usual rehabilitation regime.

The patient was discharged Sept. 8, 1953. The chest x-ray showed stability and gastric cultures were negative for acid-fast bacilli in July, 1955.

CASE 5.—L. B., 144799, a 28-year-old white man, was admitted to the hospital on Feb. 24, 1953, because of moderately advanced, active, cavitary tuberculosis of the right upper lobe. *After 11 months' therapy with streptomycin and PAS*, a right thoracotomy was performed on Feb. 2, 1954, because of persistent cavitary disease (Fig. 10).

At thoracotomy a right apical and posterior segmentectomy were performed to remove all of the palpable disease. Four days postoperatively and 2 days after removal of pleural drainage tubes, an apical pneumothorax space was noted above the fifth posterior rib. A catheter was inserted in the apical space for 10 days but the space persisted (Fig. 11) until April 26, 1954. The patient's convalescence was otherwise uneventful and check-up examination in September, 1955 (Fig. 12), revealed a stable roentgen picture and gastric cultures were negative for tubercle bacilli.

CASE 6.—J. R., 148512, a 47-year-old white man, was admitted to the hospital Aug. 19, 1953, as a transfer from another institution because of persistent cavitary, moderately advanced, active, pulmonary tuberculosis in spite of adequate antibiotic and chemotherapy for months (Fig. 13). A right thoracotomy was performed on Sept. 17, 1953, and a posterior gmentectomy of the upper lobe was performed.

The patient's immediate postoperative course was satisfactory and the pleural catheter s removed and the lung well expanded on the fourth day after operation. Ten days post- eratively, an apical pneumothorax space was found associated with right chest pain and cutaneous empyema. A catheter was reinserted at the apex of the right thorax and it nctioned for 7 days after which it was withdrawn. The space, persisted however (Fig. 14), til Nov. 2, 1953. A follow-up x-ray in June, 1954 (Fig. 15), revealed a stable picture and stric cultures in May, 1955, were negative for acid-fast bacilli.

## CONCLUSIONS

1. Residual pleural space was a complication in one third of 100 consecu- e small resections for pulmonary tuberculosis.
2. The fate of the residual space after small resections is usually one of adual resolution, and infection of this space is rare.
3. Careful expectant observation can be safely advised for the postoperative eural spaces, following small tuberculous resections, if physician and patient e confident that a regular schedule of follow-up examination will be adhered by both parties.

## REFERENCES

Chamberlain, J. M., Storey, C. F., Klopstock, R., and Daniels, C. F.: Segmental Resection for Pulmonary Tuberculosis, J. THORACIC SURG. 26: 471-485, 1953.

Douglass, R., Bosworth, E. B., Judd, J. M., and Chang, K. H.: Resection Surgery in Tuberculosis, J. THORACIC SURG. 29: 136-250, 1955.

Franz, B. J., and Murphy, J. D.: The Masked Bronchopleural Fistula, J. THORACIC SURG. 29: 512-517, 1955.

# 5 HYDROXYTRYPTAMINE (SEROTONIN) AND ITS RELATIONSHIP TO THE CARCINOID CARDIOVASCULAR SYNDROME AND RENDU OSLER WEBER'S SYNDROME

CHARLES H SPARKS, M D,* AND THOMAS L TOMBRIDGE, M D**
ST LOUIS, MO

DURING the past few years, the carcinoid cardiovascular syndrome has been recognized and described We wish to review the literature, add one case, and discuss the pathogenesis of the various features of the syndrome In addition, we will propose a new explanation for the pathogenesis of Rendu Osler Weber's syndrome (heredofamilial telangiectasia), with supporting evidence

## REVIEW OF LITERATURE

Prior to 1953, there were only sporadic reports of cases of carcinoid tumors of the intestine associated with cardiac valvular abnormalities This association was thought only to be coincidental, until Isler and Hedinger,[1] in Zurich, and Rosenbaum and his associates[36] in the United States, in 1953 independently suggested a syndromal relationship of the various components Thorson and co workers[40] in 1954, published a review and listed as essential features intestinal carcinoid tumor with liver metastasis, dependent edema diarrhea, telangiectases, asthmatic attacks, flushing and cyanosis, pulmonary stenosis, and tricuspid insufficiency The cyanosis was violaceous in color and transient, and the telangiectases were found principally on the face They listed 16 cases present in the literature up to that time including, not only those of Isler and Hedinger, and Rosenbaum, but also, 6 of their own Of these 16 cases 12 were definite and 4 were questionable to varying degrees As pointed out by McKusick[23] these writers overlooked one definite case that had appeared earlier[24] Subsequently, 9 acceptable cases have been reported,[1, 3, 17, 21] including one by McKusick[23] who wrote a comprehensive review on the subject Thus, there have been twenty one valid instances of this syndrome, to which we are adding one of our own, making a total of twenty two to date Other reports indicate possible examples, but cannot be included due to lack of autopsy confirmation or incomplete clinical study Table I includes all the cases of Thorson and associates all subsequent autopsied cases, and one case of particular interest which will be alluded to later[6] The present case is the first to have had complete ante mortem and post mortem studies, and is also the first one reported in a Negro

---

From the Departments of Thoracic Surgery and Pathology Veterans Administration Hospital St Louis Mo

Received for publication March 20 1956

*Resident in Thoracic Surgery Washington University Service Veterans Administration Hospital St Louis Mo

**Resident in Pathology St Louis University School of Medicine St Louis Mo before operation, tuberculous lesions were present in the bronchi of 67 per cent of the operation specimens. Whereas, in the group which received drugs in the immediate preoperative period, these lesions were found in only 24 per cent of the specimens.

TABLE I. EFFECT OF PREOPERATIVE ANTIBACTERIAL DRUG TREATMENT ON INCIDENCE OF TUBERCULOUS LESIONS IN THE BRONCHI

| | TOTAL NUMBER OF OPERATION SPECIMENS | WITH TUBERCULOUS LESIONS IN TRANSECTED BRONCHI |
|---|---|---|
| Patients not receiving chemotherapy before operation | 18 | 12 (67%) |
| Patients receiving chemotherapy before operation | 51 | 12 (24%) |

In Table II the incidence of these tuberculous lesions is related to the duration of chemotherapy before operation. Prolongation of drug treatment beyond one week was associated with a continued reduction in the incidence of bronchial lesions but there was little further diminution until after eighteen weeks of chemotherapy when a sharp reduction occurred.

TABLE II. INFLUENCE OF DURATION OF ANTIBACTERIAL DRUG TREATMENT ON INCIDENCE OF TUBERCULOUS LESIONS IN THE BRONCHI

| PREOPERATIVE CHEMOTHERAPY | TOTAL NUMBER OF OPERATION SPECIMENS | WITH TUBERCULOUS LESIONS IN TRANSECTED BRONCHI |
|---|---|---|
| None | 18 | 12 (67%) |
| 1 week or less | 9 | 4 (44%) |
| 1 to 9 weeks | 16 | 3 (19%) |
| 9 to 18 weeks | 15 | 4 (27%) |
| 18 to 39 weeks | 11 | 1 (9%) |

## SUMMARY AND CONCLUSIONS

The effect of preoperative chemotherapy on the histology of the bronchus has been studied in 69 patients undergoing pulmonary excision for tuberculosis.

Chemotherapy before operation appears to reduce the incidence of tuberculous lesions in the transected bronchi of operation specimens. A considerable reduction in the incidence of these lesions occurs when treatment is prolonged beyond one week, but is most obvious after eighteen weeks of chemotherapy before operation. These observations support the view expressed previously that treatment with antibacterial agents before operation reduces the incidence of bronchopleural fistula after operation by reducing the incidence of tuberculous lesions in the transected bronchi.

## REFERENCE

1. Gordon, Wally: The Effect of Preoperative Streptomycin on the Incidence of Bronchopleural Fistula After Pulmonary Excision for Tuberculosis, J. THORACIC SURG. 28: 1, 1954.

On a 2 gram salt diet, diuretics, continued digitalis, and bed rest, there was a twenty pound weight loss during a period of a few days.

Exploratory laparotomy revealed the liver at the level of the umbilicus and to contain numerous whitish tumor masses which measured 2 to 10 cm. in diameter. The mesentery of the small intestine contained numerous tumor nodules having the same appearance as those in the liver. No primary lesion was found. Biopsies were obtained from the liver and small intestinal mesentery and were found to contain carcinoid tumor.

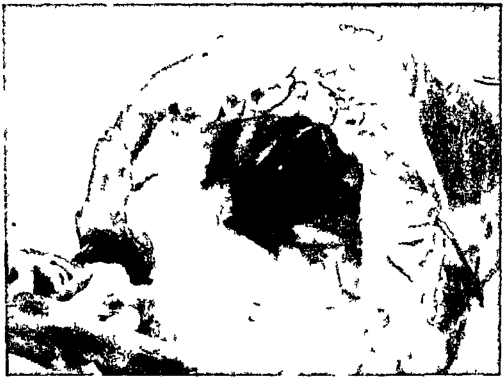


Fig. 2.—Right atrium looking through widely patent tricuspid valve orifice. Note the great endocardial thickening particularly evident on the cut edge of the atrial wall.

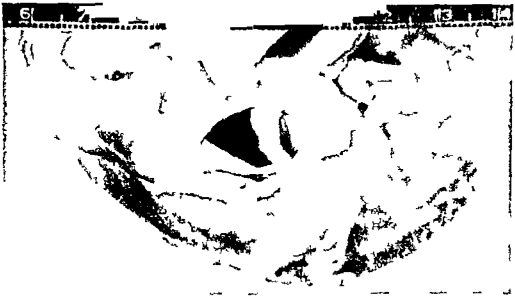


Fig. 3.—Pulmonary valve. The severe involvement of the cusps is shown here. The commissure on the left is retracted to demonstrate its fibrous fusion.

*Laboratory data.* Hemoglobin, 13.3 grams per cent, hematocrit, 47 volumes per cent sedimentation rate, 14 mm. per hour, white blood count, 5,900 cm. per milliliter, blood amylase, 133 Somogyi units, VDRL negative, and blood urea nitrogen, 19.5 mg. per cent. The stools were negative for blood, ova, and parasites. The urinalysis was normal. The serum bilirubin was 0.6 mg. per cent, alkaline phosphatase 11.0 blood urea nitrogen, thymol turbidity, 0.3, albumin, 4.0 Gm. per cent, globulin, 2.5 Gm. per cent, the Bromsulphalein retention 19 per cent after 45 minutes, and cephalin flocculation negative at 24 and 48 hours. An electrocardiogram on each admission revealed combined heart strain. Hemoglobin oxygen saturation determinations were as follows: Pulmonary artery—57.5 p

## CASE REPORT

J. B., a 33-year-old Negro male laborer, was admitted to hospital with complaints of progressive exertional dyspnea, starting three months prior to admission, and dependent edema for one week. There was no history to indicate rheumatic fever. He was well until onset of these symptoms. Examination revealed no cardiac enlargement, a Grade 2 systolic murmur was heard to the left of the sternum, $P_2$ was greater than $A_2$ and there was one-plus pitting edema of both legs. The edema disappeared after digitalization. He was discharged without a definitive diagnosis having been made and was followed at home for three months. There was gradually increasing weakness without exertional dyspnea. During the six weeks prior to the second admission, there was a constant ache in the right anterior chest and the right upper quadrant of the abdomen, occasionally accompanied by nausea. Also, during this time, he had diarrhea consisting of several watery bowel movements daily. This was controlled with paregoric and never recurred during the remainder of the course. The weight remained steady. There was no history of asthma. The patient stated that at times his face felt warm "as if it were in an oven."

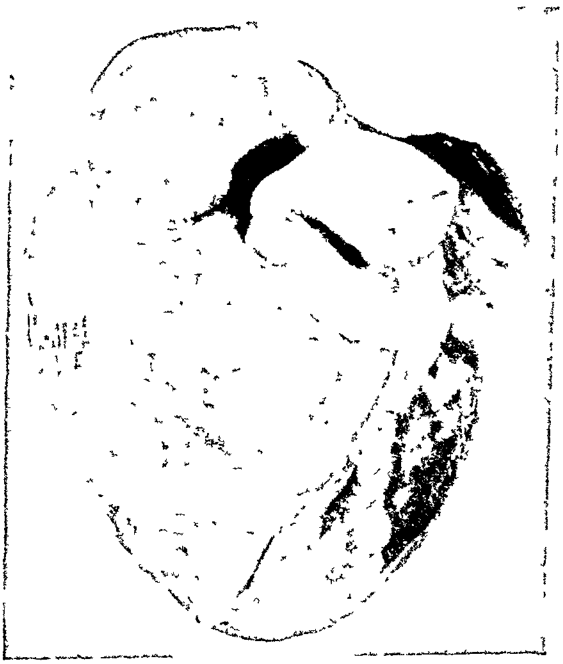


Fig. 1—External view of heart showing prominent right heart enlargement, both atrial and ventricular.

Examination at the second admission revealed an alert young Negro man, not appearing acutely or chronically ill. The skin and mucous membranes were normal. The blood pressure was 110/80 mm. Hg and the pulse was regular. There was minimal venous pulsation in the neck. There was no cardiac enlargement. Grade 2 systolic and diastolic murmurs were heard best near the left sternal border in the second and third intercostal spaces and $P_2$ was greater than $A_2$. The lungs were clear. The liver was palpable at the level of the umbilicus and was firm, nodular, and moderately tender without pulsation. Examination of the lower extremities revealed one-plus pitting edema to the mid-leg, bilaterally. Cardiac catheterization was performed establishing diagnoses of pulmonary hypertension, pulmonary insufficiency, and tricuspid insufficiency. Hemoglobin oxygen saturation determinations revealed no left-to-right shunt. A Vim-Silverman needle biopsy of the liver contained carcinoid tumor.

lower lobe of the right lung, the remaining findings were limited to the heart. The heart weighed 300 Gm. There was obvious right heart enlargement, both atrial and ventricular. The pulmonary valve admitted the tip of the little finger with difficulty, and the valve cusps were markedly fibrotic and stubby in appearance, with fusion of the commissures. The entire right atrium, including the auricular appendage, was rigid and showed a glistening, white, very firm endocardium which varied between 1 and 2 mm. in thickness. Its consistency on cutting was noted to be more like that of cartilage than fibrous connective tissue. This process ended rather abruptly at the tricuspid valve, with only minimal, patchy involvement of the right ventricular endocardium. The tricuspid valve, however, had severely sclerotic leaflets which were thickened and retracted. The chordae tendineae were very short and thick, those of the septal cusp being only a few millimeters in length. As a result, there was an abnormally wide patency of the valve orifice with obvious incompetency. The right ventricle was moderately hypertrophied, measuring 5 mm. in thickness The mitral valve leaflets had only slight thickening at their free edges, and the chordae tendineae were very minimally affected, except for one from the anterior leaflet, which was considerably shortened. The left atrium, in contrast to the right, was soft, pliable, and of normal size. The endocardium did appear somewhat thickened, however. The left ventricle and aortic valve were entirely normal.

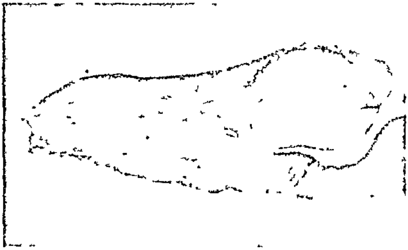


Fig 6—Low-power view of a pulmonary valve cusp which has been converted into a mere protuberant nodule of fibrous tissue There is a complete lack of involvement of the pulmonary artery (on the right) or the right ventricular endocardium (left)

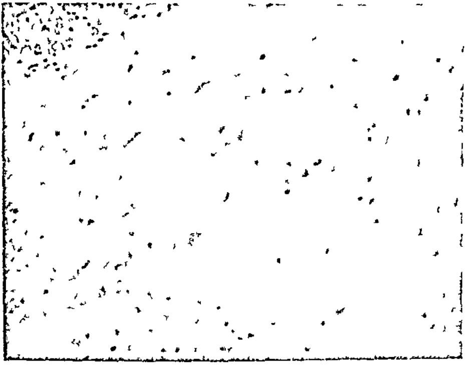


Fig 7—Right atrial endocardium under medium power, myocardium in upper left corner. The sclerosis as seen here, is of a relatively acellular, fibrillar type. Note the absence of inflammatory infiltrate

cent, right ventricle—54.9 per cent, right atrium—53.2 per cent, and femoral artery—91.3 per cent. Qaulitative test for 5-hydroxy-indol-acetic acid in the urine was strongly positive.[38] Chest x-rays made on both admissions were normal and revealed no total or chamber cardiac enlargement.

The course was slowly downhill and the patient died fifteen months after the initial onset of symptoms. During the last three months there was increasing congestive heart failure not responding to therapy and intermittent small bowel obstruction requiring intubation.

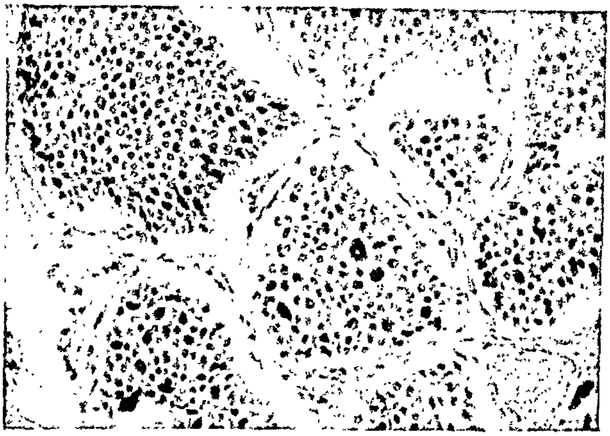


Fig. 4.—Medium high-power view of metastasis in liver. This pattern is typical of carcinoid tumors. As mentioned in the text, there is a fairly marked variation in nuclear size.

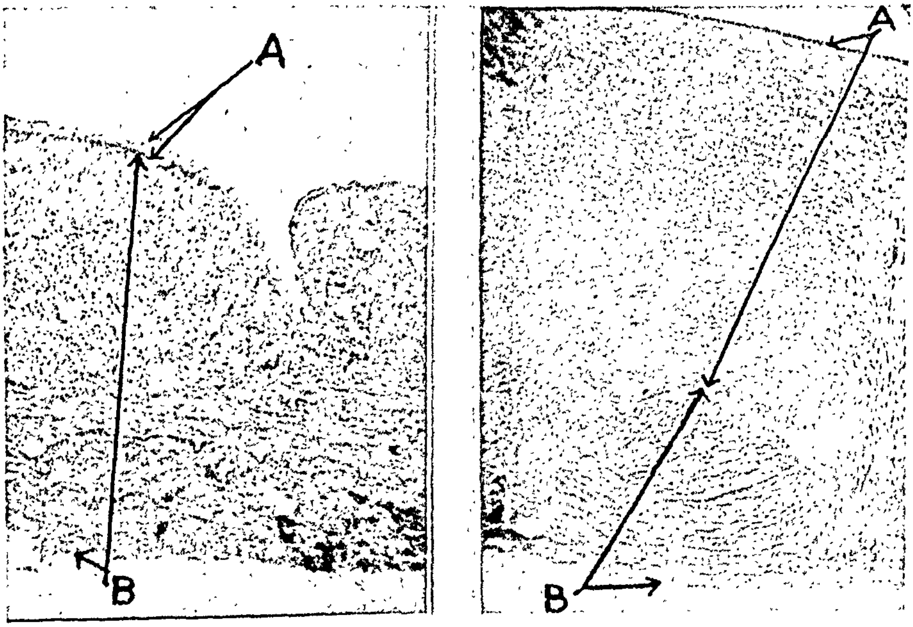


Fig. 5.—Normal atrial wall (left) as compared with the right atrium (right) in this case. *A*, endocardium; *B*, myocardium. The extreme endocardial thickening is obvious.

*Pathologic findings:* At autopsy, the peritoneal cavity contained many fibrous adhesions resulting in kinking of the small intestine. A small (1½ cm.), primary carcinoid tumor was found in the ileum, with local mesenteric and para-aortic lymph node involvement. There was no peritoneal involvement. The liver contained many large yellowish-white tumor nodules and weighed 1,800 Gm. Except for some focal parenchymal hemorrhage in the counterpart in the evacuation of the bowels noted in dogs as well as the subjective desire for a bowel movement in man. "Asthmatic" attacks, or transient bronchial constriction, have been observed in cats by Comroe,[4] and similar effects were noted in rabbits and guinea pigs by Freyburger and his associates.[11] The cardiac changes at present are entirely unexplained, although Thorson and co-workers,[40] and McKusick[23] have speculated on them. That there is some direct effect of 5-HT on the endocardium appears certain, in view of the key case reported by McKusick which showed marked left heart involvement in the presence of a patent foramen ovale. It has been shown by Page,[26] and others[2, 18, 29] that 5-HT is detoxified in the lungs, explaining why such an abnormal opening is necessary for left heart involvement. In regard to the flushing phenomenon, Reid[32] produced a red reaction without itching or whealing in human skin by intracutaneous injection. This red area was surrounded by a zone of venoconstriction. In a patient, still living, reported by Daugherty and co-workers[6] manual pressure on a proved carcinoid tumor produced, after 90 seconds, a warm sensation followed by a red blush that lasted for one minute. Serum serotonin level determined during a period of *nonflushing* was zero (0), while during a flushing episode it was 167 mcg. per 100 ml. of serum. This certainly indicates a causal relationship between the 5-HT released by the tumor and the flushing phenomenon.

In considering all of the cases of this syndrome which are included in Table I, it is immediately apparent that the cardiovascular components present themselves in the following observed combinations: (1) right heart changes, plus left heart changes, plus cutaneous phenomena, (2) cutaneous phenomena, (3) right heart changes, and (4) right heart changes plus cutaneous phenomena.

Cases 9, 22, 23, and 26 represent four examples of combination No. 1. In Case 26, there was found at autopsy a patent foramen ovale. It can be concluded that 5-HT, in order to escape detoxification, must by-pass the lungs enroute to the left heart, or exceed the detoxifying capacity of the lungs. One can then, in theory, assume that in Cases 9, 22, and 23, 5-HT also by-passed the lungs. Evidence will be cited later to support the view that clinically unrecognized pulmonary arteriovenous fistulas represent the route of by-pass. Case 20 represents an example of combination No. 2 and would suggest that the 5-HT level is well below the detoxifying capacity of the lungs, since there were no right heart changes. Then again, it is suggested that 5-HT by-passed the lungs in order to get into the arterial circulation in its active form to produce the cutaneous phenomena. Cases 7 and 8 are examples of combination No. 3. In these cases one can theorize that there is no means for 5-HT to bypass the lungs, that there was sufficient 5-HT in the blood returning to the heart to produce the right heart changes but still within the detoxifying capacity of the lungs, since there were no left heart changes and there was absence of cutaneous phenomena, i.e., flushing and telangiectasia. In carrying *this same type of reasoning further, one would conclude that in patients corresponding* to combination No. 4, the 5-HT detoxifying capacity of the lungs was exceeded enough to deliver a sufficient amount to pr[illegible] th[illegible]taneous phenomena, but insufficient to produce the left heart c[illegible] then

Microscopically, the primary tumor and its metastases demonstrated a typical carcinoid pattern, except for increased nuclear pleomorphism, possibly indicating a higher degree of malignancy, although this feature has been noted in many types of endocrine tumors. Sections from the right atrium showed a relatively acellular, poorly vascularized endocardial thickening with no inflammatory infiltrate present. Changes in the tricuspid valve were entirely similar, again showing no vascularization or inflammation. The pulmonic valve, however, did contain an increased number of blood vessels and lymphocytes near its ventricular surface. Histochemically, the abundant ground substance in the right atrial endocardium was strongly metachromatic with toluidine blue, while the delicate fibrils present took a positive Masson's trichrome stain for connective tissue. The metachromasia was reversible with testicular hyaluronidase, giving the sclerotic endocardium a very close histochemical resemblance to cartilage. This is interesting in view of the gross similarity to cartilage in appearance and consistency (see above). There was a conspicuous lack of the "collagen scarring" and vascularization mentioned by Thorson and associates,[40] as well as absence of the marked inflammatory infiltrate present in McKusick's case.[23] In general, the histologic changes here appear distinctive, and do not seem to be those of a "burnt out endocarditis"[10] or a primary inflammatory process.

*Summary of Anatomic Diagnoses.*—

A. Carcinoid tumor of the ileum with hepatic and local lymph node metastases.

B. Endocardial sclerosis of right atrium and pulmonary and tricuspid valves, with pulmonic stenosis and tricuspid insufficiency. Minimal thickening of mitral valve leaflets and their chordae tendineae. Right heart dilatation and hypertrophy.

C. Peritoneal adhesions and kinking of small intestine.

## DISCUSSION

It has been known for many years that a vasoconstrictor substance is present in the serum after blood has clotted.[20, 39] The pharmacology of this material has been studied extensively in recent years, and has been isolated in pure form by Rapport,[30] and given the name "serotonin." This substance was subsequently synthesized by Hamlin and Fisher,[16] in 1951, and is now known to be 5 OH tryptamine (5-HT). Experimental evidence indicates that the bulk of serotonin in the blood is carried in platelets but is not formed there.[12, 33]

The source of serotonin, however, was discovered by an entirely different method of investigation. Long ago, Masson[15] considered the enterochromaffin system as a diffuse endocrine organ analogous to the pancreatic islets, and carcinoid tumors, which arise from these chromaffin cells, were held by him to be endocrine tumors. Erspamer,[7, 9, 10] in a long series of experimental studies on this subject, eventually extracted a hormonally active material from gastroenteric mucosa, which he named "enteramine." Later, he and Asero[8] showed this substance to be 5-HT, thus chemically identical to serotonin. Lembeck,[22] in 1953, extracted large amounts of 5-HT from a carcinoid tumor, confirming the speculation of Masson forty years previously. The exact physiologic role or roles of 5-HT in the human body has not been determined conclusively; however, the effects of administration have been studied in man and several species of animals by Page and co-workers,[27, 28] Comroe,[4] Reid,[32, 34, 35] Correll,[5] and Freyburger[11] and their associates, Erspamer,[7, 9, 10] and others. Except for the cardiac lesions, the main features of the syndrome are easily explained in the light of this experimental work on serotonin effects. The diarrhea finds its

## RENDU OSLER WEBER'S SYNDROME

Rendu Osler-Weber's syndrome (heredofamilial telangiectasia) is defined as telangiectasia of the skin and mucous membranes which is occasionally fatal, due to hemorrhage. Garland and Anning[13] reviewed the literature on this subject and presented voluminous evidence supporting the hereditary factor in this syndrome.

As mentioned above, Goldman attempted to establish pulmonary A-V fistula as a manifestation of Rendu Osler Weber's syndrome, i.e., telangiectasia of the lungs. We wish to challenge this concept in Rendu Osler-Weber's syndrome and to offer a new explanation for its etiology with supporting evidence.

The finding by Muir of telangiectasia (Rendu Osler Weber's syndrome) in 53 per cent of 117 cases of proved pulmonary A V fistula proves a more than coincidental relationship between the two entities. Yater,[40] Shefts,[3] Whitaker,[41] and others support this view by stating that pulmonary A-V fistula should be considered a part of Rendu Osler Weber's syndrome and cite supporting evidence for this statement.

We wish to offer the hypothesis that the telangiectasis in Rendu Osler Weber's syndrome is produced by 5 HT which escapes detoxification by bypassing the lungs via a pulmonary A-V fistula (or fistulas) which may or may not be demonstrable clinically or roentgenographically. Data reported by Daugherty and co-workers[6] and others establish that 5 HT is a normal constituent of the blood and is simply increased in patients who have the carcinoid cardiovascular syndrome. There is conclusive evidence that pulmonary A V fistulas exist which cannot be diagnosed clinically or roentgenographically. The authors are conducting studies on consecutive routine autopsy cases to determine this incidence.

The foregoing evidence would indicate that one should find telangiectasia in patients with intracardiac right-to left shunts. In a personal communication Dr Chester P Lynxwiler, St Louis, Mo, stated that telangiectasia is not uncommonly found developing in patients with the tetralogy of Fallot who survive to the second decade and longer. The authors are investigating this incidence further.

## CONCLUSIONS

1 It would seem that the concentration of 5 HT reaching the heart chambers must be greater to produce the endocardial changes than that required for the cutaneous phenomena.

2 The length of time of exposure to 5 HT is obviously a factor in the production of the endocardial changes and telangiectasis, but not the flushing. In view of the fact that, in the reported cases of carcinoid cardiovascular syndrome, there were several cases of telangiectasia without left heart changes but none with left heart changes and no telangiectasia, one can conclude that with a given concentration of 5 HT in the arterial circulation, a longer time will be required to produce the left heart changes than to produce telangiectasia.

3 The telangiectasia commonly seen associated with pulmonary A V fistula is probably due to 5 HT escaping detoxification in t[illegible]ng through the fistulas.

follow that a greater concentration of 5-HT in the blood reaching the systemic arterial circulation is required to effect the left heart changes than that required to produce telangiectasis.

In carrying out the above analysis, information from an entirely different source took on particular significance. In 1955, Muri[25] reviewed the literature and found 117 reported cases of pulmonary arteriovenous (A-V) fistula. Sixty-two (53 per cent) of these patients were reported to have telangiectasia. The other 55 may or may not have had it as the presence or absence was not reported. Muri mentioned that some of these patients had episodes of reddish-blue cyanosis.

TABLE I

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25 | 26 | 27 | 28 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| SEX | M | M | F | F | M | F | F | F | M | M | M | M | M | M | — | — | F | M | M | F | F | F | M | F | F | M | F | M |
| AGE AT DEATH | 19 | 63 | — | — | 43 | 59 | 56 | 64 | 33 | 47 | 43 | 32 | 31 | 48 | — | — | 44 | 76 | 40 | 63 | 59 | 42 | 43 | 68 | 65 | 68 | — | 33 |
| INT. CARCINOID | + | + | + | + | + | + | + | + | + | + | ? | ? | ? | ? | + | 0 | + | + | + | + | + | + | + | + | + | + | + | + |
| LIVER METAS. | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | − | + |
| OVARIAN METAS. | 0 | 0 | + | + | 0 | + | + | + | 0 | 0 | 0 | 0 | 0 | 0 | − | − | 0 | 0 | 0 | + | − | − | 0 | − | − | + | + | 0 |
| PULM. STEN. | + | + | ? | 0 | ? | ? | + | + | + | + | + | + | + | − | + | + | + | + | + | 0 | + | + | + | − | − | + | − | 0 |
| TRICUSP. INSUFF. | + | 0 | ? | ? | ? | 0 | + | + | + | + | 0 | − | 0 | − | ST. | ST. | ST | − | + | 0 | + | + | + | + | + | + | − | + |
| ASTHMA | + | + | + | 0 | − | − | − | − | − | − | − | ? | + | − | − | − | − | − | − | − | − | − | − | − | − | − | − | 0 |
| AUTOPSY | + | + | L | L | NO | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | L | + |
| AORTIC DISEASE | 0 | 0 | ? | ? | ? | 0 | 0 | 0 | + | 0 | 0 | 0 | 0 | − | 0 | 0 | 0 | 0 | − | 0 | 0 | + | + | − | − | + | − | 0 |
| MITRAL DISEASE | 0 | 0 | ? | ? | ? | 0 | 0 | 0 | + | 0 | 0 | 0 | 0 | − | + | 0 | 0 | 0 | − | 0 | 0 | + | + | + | + | + | − | + |
| EDEMA | + | + | 0 | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | 0 | 0 | + |
| REFERENCE NO. | $T_1$ | $T_2$ | $T_3$ | $T_4$ | $T_5$ | $T_6$ | $T_7$ | $T_8$ | $T_9$ | $T_{10}$ | $T_{11}$ | $T_{12}$ | $T_{13}$ | $T_{14}$ | $T_{15}$ | $T_{16}$ | 24 | 17 | 3 | 21 | 21 | 1 | 1 | 31 | 31 | 23 | 6 | A |
| CYANOSIS | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | − | + | − | − | + | + | − | + |
| FLUSH OR PLETHORIC COLORATION | + | + | + | + | + | + | − | − | + | + | + | + | + | + | + | + | + | + | + | + | + | + | + | − | + | + | + | + |
| RIGHT ATRIAL PRESSURE (mmHg) | HIGH | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | 58/24 |
| DIARRHEA | + | + | + | + | − | + | + | + | 0 | + | + | + | + | 0 | − | − | − | + | + | + | + | + | − | − | + | + | + | + |
| 5 HIAA (URINE) | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | − | + | + |
| TELANGIECTASIS | + | − | − | − | − | − | − | − | − | − | − | − | − | + | + | + | − | − | + | − | − | + | − | − | − | + | 0 | 0 |

L = living.
+ = proved.
? = possible but not proved.
− = neither proved nor disproved.
0 = proved absence.
St. = stenosis.
$T_1$–$T_{16}$ = cases reported by Thorson, et al.[40]
5 HIAA = 5-hydroxy-indol-acetic acid.

Goldman[14] considers pulmonary A-V fistula to be a manifestation of Rendu-Osler-Weber's syndrome, i.e., a pulmonic manifestation of the telangiectasia. In view of the part played by 5-HT in the carcinoid-cardiovascular syndrome, it is proposed that 5-HT, which is known to be produced normally by the enterochromaffin system,[8] by-passes the lungs by way of the pulmonary A-V fistulas in sufficient quantity to produce the telangiectasia. We might then expand this reasoning to hypothesize that Rendu-Osler-Weber's syndrome is a result of pulmonary A-V fistulas, whether or not they are clinically demonstrable.

10 Erspamer, V Pharmacology of Indolealkylamines, Pharmacol Rev 6 525, 1954

11 Freyburger, W A, Graham, B L, Rapport, M M, Seay, P H, Govier, W M, Swoap, O F, and Van der Brook, M J The Pharmacology of 5 Hydroxytryptamine (Serotonin), J Pharm & Exper Therap 105 80, 1952

12 Friedman, B, Rapport, M M, and Zucker, M B Identification of Serotonin in Blood Platelets, Proc Soc Exper Biol & Med 85 1954

13 Garland, H G, and Anning, S T Hereditary Haemorrhagic Telangiectasia A Genetic and Bibliographical Study, Brit J Derm & Syph 62 289, 1950

14 Goldman, A Arteriovenous Fistula of the Lung, Am Rev Tuberc 57 266, 1948

15 Gosset, A, and Masson, P Tumeurs endocrines de l'appendice, Presse méd 22 237, 1914

16 Hamlin, K E, and Fisher, F E Synthesis of 5 Hydroxytryptamine, J Am Chem Soc 73 5007, 1951

17 Hedinger, C, and Gloor, R Metastierende Dunndarmkarzinoide, tricuspidalklappenveranderungen und pulmonalstenose ein neues syndrome, Schweiz med Wchnschr 84 942, 1954

18 Herrick, J, and Markowitz, J The Toxic Effects of Defibrinated Blood When Perfused Through the Isolated Mammalian Heart, Am J Physiol 88 698, 1929

19 Isler, P, and Hedinger, C Metastasierendes Dunndarmkarcinoid mit schweren vorwiegend das rechte Herz betreffenden Klappenfehlern und Pulmonalstenose—ein eigenartiger Symptomenkomplex? Schweiz med Wchnschr 83 4, 1953

20 Janeway, T C, Richardson, H B, and Park, E A Experiments on the Vasoconstrictor Action of Blood Serum, Arch Int Med 21 565, 1918

21 Jenkins, J S, and Butcher, P J A Malignant Argentaffinoma With Cyanosis and Pulmonary Stenosis, Lancet 1 331, 1955

22 Lembeck, F 5 Hydroxytryptamine in a Carcinoid Tumor, Nature 172 910, 1953

23 McKusick, V A Carcinoid Cardiovascular Disease, Bull Johns Hopkins Hosp 98 13, 1956

24 Millman, S Tricuspid Stenosis and Pulmonary Stenosis Complicating Carcinoid of the Intestine With Metastases to the Liver, Am Heart J 25 391, 1943

25 Muri, J W Arteriovenous Aneurysm of the Lung, Am J Surg 89 265, 1955

26 Page, I H Serotonin (5 Hydroxytryptamine), Physiol Rev 34 563, 1954

27 Page, E W, and Glendening, M B Production of Renal Cortical Necrosis With Serotonin, Am J Med 19 285, 1955

28 Page, I H, and McCubbin, J W The Variable Arterial Pressure Response to Serotonin in Laboratory Animals and Man, Circulation Res 1 354, 1953

29 Rapport, M M, Green, A A, and Page, I H Enzymatic Inactivation of Serum Vasoconstrictor, Proc Soc Exper Biol & Med 68 582, 1948

30 Rapport, M M Serum Vasoconstrictor (Serotonin) V Presence of Creatinine in the Complex A Proposed Structure of the Vasoconstrictor Principle, J Biol Chem 180 961, 1949

31 Ratzenhofer, M, and Lembeck, F Über den Gehalt an 5 Oxytryptamin in Karcinoiden des Darmtraktes, Ztschr Krebsforsch 60 169, 1954

32 Reid, G Physiological Effects of Partially Purified Serum Vasoconstrictor (Serotonin), Australian J Exper Biol & M Sc 29 401, 1951

33 Reid, G, and Bick, M Pharmacologically Active Substance in Serum, Australian J Exper Biol & M Sc 29 401, 1951

34. Reid, G Circulatory Effects of 5 Hydroxytryptamine, J Physiol 118 435, 1952

35 Reid, G Pharmacological Actions of 5 Hydroxytryptamine (Serotonin), Thrombocytin, Nature 169 801, 1952

36 Rosenbaum, F F, Santer, D G, and Claudon, D B Essential Telangiectasia, Pulmonary Stenosis and Neoplastic Liver Disease A Possible New Clinical Syndrome (Abstract), J Lab & Clin Med 42 941, 1953

37 Shefts, L M Discussion of Maier, Himmelstein, Riley, and Bunin, J THORACIC SURG 17 13, 1948

38 Sjoerdsma, A, Weissbach, H, and Udenfriends, S Simple Test for Diagnosis of Metastatic Carcinoid (Argentaffinoma), J A M A 159 397, 1955

39 Stevens, L T, and Lee, F S Action Intermittent Pressure and of Defibrinated Blood Upon Blood Vessels of Frog and Terrapin, Johns Hopkins Biol Studies 3 99, 1884

40 Thorson, A, Biorck, G, Bjorkman, G, and Walderstrom, J Malignant Carcinoid of the Small Intestine With Metastases to the Liver, Valvular Disease of the Right Side of the Heart (Pulmonary Stenosis and Tricuspid Regurgitation, Without Septal Defect), Peripheral Vasomotor Symptoms, Bronchoconstriction, and an Unusual Type of Cyanosis A Clinical and Pathological Syndrome, Am Heart J 47 795, 1954

41 Whitaker, W Cavernous Haemangioma of the Lung, Thorax 2 58, 1947

42 Yater, W M, Finnegan, J, and Giffin, H M Pulmonary Arteriovenous Fistula (Varix), J A M A 141 581, 1949

4. Pulmonary A-V fistulas, which are not necessarily demonstrable clinically or roentgenographically, are probably the route taken by 5-HT in bypassing the lungs and therefore escaping detoxification to produce the telangiectasia in Rendu-Osler-Weber's syndrome. If this is true, Rendu-Osler-Weber's syndrome is a result of pulmonary A-V fistulas. In this event, the great amount of evidence in the literature to support the view that Rendu-Osler-Weber's syndrome is an inherited characteristic then becomes evidence to support pulmonary A-V fistula as the inherited characteristic. Further investigative work must be carried out to confirm this hypothesis. Cases of Rendu-Osler-Weber's syndrome should be investigated for increased blood levels of 5-HT and presence of pulmonary A-V fistula. Therefore, it is recommended that in these cases chest x-rays be made, blood hemoglobin oxygen saturation be determined, and special procedures be carried out on autopsy material in search for pulmonary A-V fistulas not demonstrable clinically.

5. In all cases of proved pulmonary A-V fistula, but especially those with telangiectasia, levels of 5-HT should be determined before and after the fistula is eradicated by surgery. At the same time, arterial hemoglobin oxygen saturations should be determined before and after surgery and compared to the changes found in the 5-HT levels.

## SUMMARY

1. A review of the literature of the carcinoid-cardiovascular syndrome is presented and one case added.

2. The various features of the carcinoid-cardiovascular syndrome are discussed with particular emphasis on the probable role of right-to-left shunts in the evolution of the cardiovascular phenomena.

3. An explanation for the telangiectasia seen in a large per cent of clinically demonstrable pulmonary A-V fistulas is offered with supporting evidence.

4. The accepted hereditary factor in Rendu-Osler-Weber's syndrome s challenged and a new explanation for its pathogenesis offered.

5. Investigative procedures for patients with Rendu-Osler-Weber's syndrome and with clinically demonstrable pulmonary A-V fistulas are recommended.

## REFERENCES

1. Bean, W. B., Olch, D., and Weinberg, H. B.: The Syndrome of Carcinoid and Acquired Valve Lesions of the Right Side of the Heart, Circulation 12: 1, 1955.
2. Bradley, T. R., Butterworth, R. F., Reid, G., and Trautner, E. M.: Nature of Lung Enzyme Which Inactivates Serum Vasoconstrictor, Nature, London 166: 911, 1950.
3. Branwood, A. W., and Bain, A. D.: Carcinoid Tumor of the Small Intestine With Liver Metastases, Pulmonary Stenosis and Atypical Cyanosis, Lancet 2: 1259, 1954.
4. Comroe, J. H., Jr.: Direct and Reflex Cardiopulmonary Effects of Serotonin (5-Hydroxytryptamine)—Their Relation to Pulmonary Embolism, Am. J. Physiol. 171: 715, 1952.
5. Correll, J. T., Lyth, L. F., Long, S., and Vanderpoel, J. C.: Some Physiologic Responses to 5-Hydroxytryptamine Creatinine Sulfate, Am. J. Physiol. 169: 537, 1952.
6. Daugherty, G. W., Manger, W. M., Roth, G. M., Flock, E. V., Childs, D. S., Jr., and Waugh, J. M.: Malignant Carcinoid With Hyperserotonemia Occurring Spontaneously or Induced by Palpation of the Tumor or by Intravenous Histamine: Report of a Case, Proc. Staff Meet., Mayo Clin. 30: 595, 1956.
7. Erspamer, V.: Die enterochromaffin Zellen der Gallenwege in normalen und pathologischen Zustanden, Virchows Arch. path. Anat. 297: 70, 1936.
8. Erspamer, V., and Asero, B.: Identification of Enteramine, the Specific Hormone of the Enterochromaffin Cell System, as *5-Hydroxytryptamine*, Nature 169: 800, 1952.
9. Erspamer, V.: Observations on the Metabolism of Endogenous 5-Hydroxytryptamine (Enteramine) in the Rat, Experimentia 10: 471, 1954.

In Part 3, the peripheral vascular response to hyperventilation in patients whose venous blood had been "arterialized" was studied by the impedance plethysmograph.

### METHODS AND CLINICAL MATERIAL

The "arterialization" of venous blood was accomplished by the method of Werner and Cooper,[1] except that the forearm was immersed in water at a temperature of 44° to 46° C. for 20 instead of 10 minutes. A thermostatically controlled water bath was employed. The $pO_2$ and $pCO_2$ were determined by the method of Riley[6] with the Scholander blood gas tension apparatus. Thirty-unit Roughton-Scholander syringes were used. All tension determinations were accomplished in duplicate and mean values reported. The mean differences between duplicate determinations were 2.3 mm. Hg for $pO_2$ and 2.4 mm. Hg for $pCO_2$.

It was found that a number of patients, with the forearm immersed in the water bath, complained of faintness after completing 20 seconds of maximum deep breathing, the method employed by Bjork. The same patients did not have that problem after taking five deep breaths (average time, 16.8 seconds). It was for that reason that hyperventilation consisting of five deep breaths taken at the patient's own speed was employed in Parts 1 and 2. In Part 3, the responses of the peripheral vascular system to the two methods of hyperventilation were studied.

All subjects were male adults and patients at the Veterans Administration Hospital, Providence, R. I.

In Part 1, 20 patients were studied. They had no clinical evidence of pulmonary or cardiovascular disease. The 8 patients studied in Part 2 had no evidence of peripheral vascular disease, but did demonstrate varying degrees of pulmonary dysfunction. The established clinical diagnoses on these patients are presented in Table I. Arterial blood was obtained from the brachial artery 24 hours after the "arterialized" venous blood samples because the intricacies of blood gas tension analysis techniques precluded simultaneous examination of the number of arterial and "arterialized" venous blood samples necessary to make the comparisons with duplicate determinations. The 18 patients studied in Part 3 had no clinical evidence of pulmonary or cardiovascular disease.

TABLE I.—ESTABLISHED DIAGNOSIS OF PULMONARY DISEASE—8 PATIENTS IN WHOM THE $pO_2$ AND pCO OF "ARTERIALIZED" VENOUS BLOOD AND ARTERIAL BLOOD BEFORE AND AFTER HYPERVENTILATION WERE COMPARED

| PATIENT | AGE YR. | PULMONARY DISEASE |
|---|---|---|
| a | 62 | Diffuse pulmonary metastases from carcinoma of prostate* |
| b | 47 | Left pneumonectomy, and thoracoplasty, postoperative for carcinoma of lung† |
| c | 32 | Right middle lobectomy, postoperative, for bronchiectasis, bronchiectasis, left lower lobe |
| d | 58 | Right pneumonectomy, postoperative, for carcinoma of lung |
| e | 59 | Chronic bronchitis and emphysema |
| f | 61 | Chronic bronchitis and emphysema* |
| g | 34 | Postoperative decortication for pyopneumothorax, right* |
| h | 59 | Silicotuberculosis, bilateral |

*Dyspnea at rest.
†Dyspnea on exertion.

# THE USE OF "ARTERIALIZED" VENOUS BLOOD FOR THE DETERMINATION OF CHANGES IN THE ARTERIAL $O_2$ AND $CO_2$ TENSIONS FOLLOWING HYPERVENTILATION

DAVID L. BROOK, M.D.,* JAMES B. KNIGHT, JR., M.D.,**
AND PHILIP COOPER, M.D.***
PROVIDENCE, R. I.

IN a previous publication,[1] it was reported that the $O_2$ and $CO_2$ tensions of "arterialized" venous blood approximated those of arterial blood in patients, at rest, with *no clinical evidence of peripheral* vascular disease. That study was the first part of an over-all evaluation of the use of "arterialized" venous blood as a substitute for arterial blood in determining changes in the $pO_2$ and $pCO_2$ of arterial blood following hyperventilation or exercise in normal subjects and in patients with or without cardiopulmonary disease. Björk[2] had reported on the change in $pO_2$ and $pCO_2$ of arterial blood following hyperventilation and exercise. If "arterialized" venous blood could be substituted for arterial blood for the determination of these tensions, it would obviate the need for arterial punctures which may be more difficult to perform and which may involve more of a hazard to the patient.

It has been reported that hyperventilation causes peripheral vasoconstriction.[3-5] Plethysmographic studies were, therefore, accomplished before, during, and after hyperventilation on a number of patients whose venous blood had been "arterialized" to determine to what extent hyperventilation interferes with the "arterialization" process.

The present investigation was divided into three parts. In Part 1, the $pO_2$ and $pCO_2$ changes in "arterialized" venous blood after hyperventilation were studied.

Part 2 concerned patients with pulmonary disease. The purpose of that study was to compare changes in gas tensions of arterial and "arterialized" venous blood after hyperventilation. No attempt was made to correlate the results of the blood gas determinations with abnormalities of pulmonary function. The results of such a study on a larger series of patients will be reported at a later date.

---

From the Surgical Service and the Surgical Research Laboratory of the Veterans Administration Hospital, Providence, R. I., and the Department of Surgery of the Boston University School of Medicine, Boston, Mass.

Received for publication March 23, 1956.

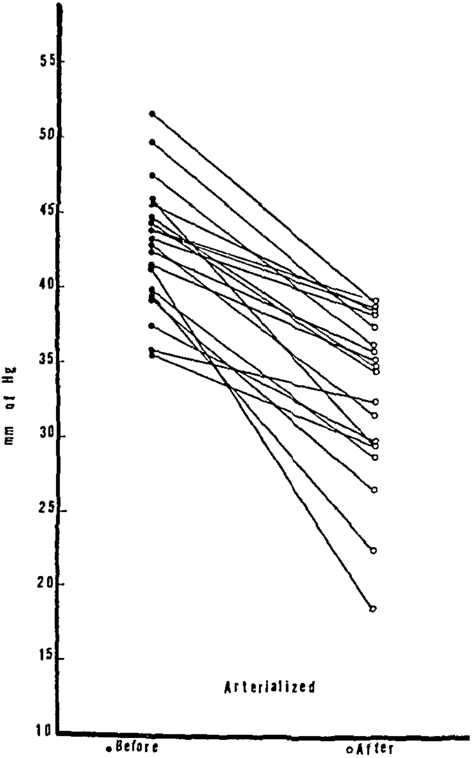
*Resident in Surgery, Veterans Administration Hospital, Providence, R. I., and Massachusetts Memorial Hospitals, Boston, Mass.

**Resident in Surgery, Veterans Administration Hospital, Providence, R. I., and Massachusetts Memorial Hospitals, Boston, Mass.

***Chief, Surgical Service, Veterans Administration Hospital, Providence, R. I., and Associate Professor of Clinical Surgery, Boston University School of Medicine, Boston, Mass.

## RESULTS

The effect of hyperventilation on the $pO_2$ and $pCO_2$ of “arterialized” venous blood (Part 1) on 20 male patients with no clinical evidence of pulmonary or cardiovascular disease is shown in Table II, and graphically presented in Figs 1 and 2 Hyperventilation produced a significant increase in $pO_2$ (mean change, +10 4 ± 1 1), and a significant decrease in $pCO_2$ (mean change, -9 7 ± 1 1 mm Hg)



$pCO_2$ BEFORE & AFTER 5 DEEP BREATHS

Fig 2—The $pCO_2$ of arterialized venous blood before and after five deep breaths—20 patients with no pulmonary or cardiovascular disease

The results of Part 2 of the study, comparing $pO_2$ and $pCO_2$ of arterial and “arterialized” venous blood following hyperventilation, are shown in Tables III, IV, V, and VI, and graphically presented in Figs 3 and 4 Hyperventilation produced a significant increase in $pO_2$ in both “arterialized” venous blood

Plethysmographic tracings were taken from the right index finger before, during, and after five deep breaths, and before, during, and after a 20-second period of maximal deep breathing, while the right forearm of each patient was immersed in a water bath for "arterialization." The impedance plethysmograph employed was fabricated in this Laboratory and is a modification of one described by Dubois and Nims.[7]

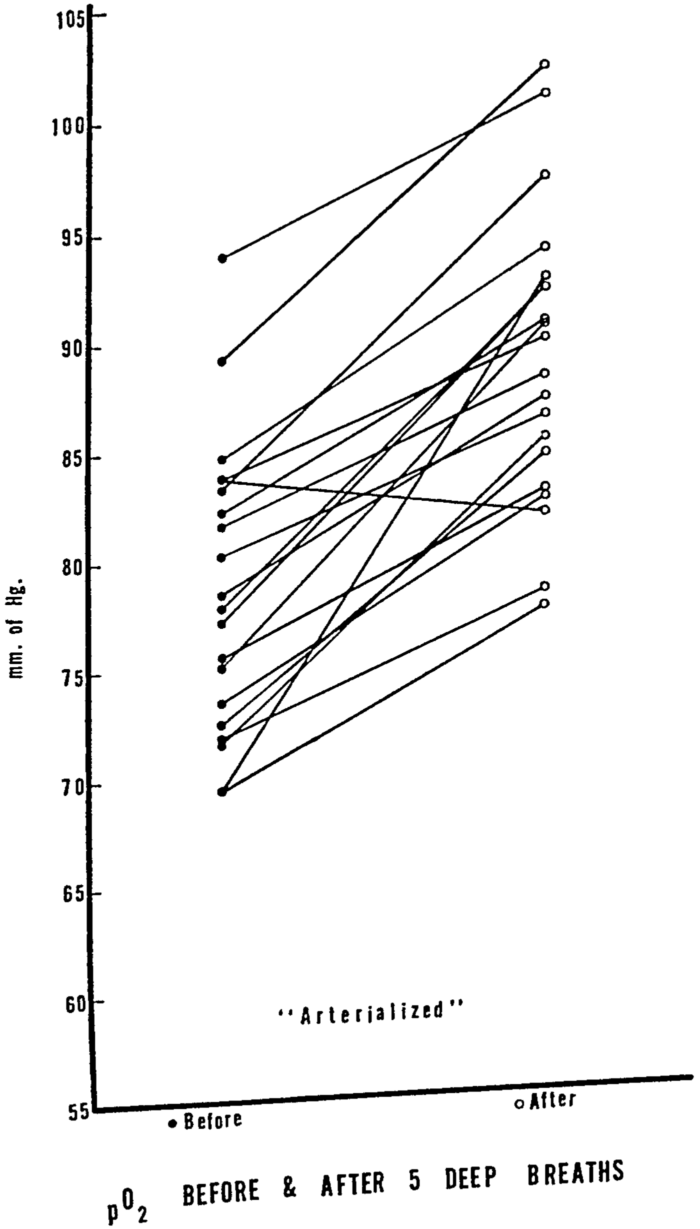


Fig. 1.—The $pO_2$ of "arterialized" venous blood before and after five deep breaths—20 patients with no pulmonary or cardiovascular disease.

All data were statistically analyzed, using the "t" test of Fisher, and a P value of 0.05 or less was considered significant.

TABLE IV DATA ON THE EFFECT OF HYPERVENTILATION ON THE $pO_2$ AND $pCO_2$ OF ARTERIAL BLOOD—PATIENTS WITH PULMONARY DISEASE

| PATIENT | $pO_2$ MM Hg |  |  | $pCO_2$ MM Hg |  |  |
|---|---|---|---|---|---|---|
|  | BEFORE | AFTER | DIFFERENCE | BEFORE | AFTER | DIFFERENCE |
| a | 54.1 | 53.2 | − 0.9* | 30.6 | 29.4 | − 1.2 |
| b | 96.2 | 99.6 | + 3.4 | 42.1 | 35.5 | − 6.6 |
| c | 87.9 | 102.8 | +14.9 | 41.7 | 31.4 | −10.3 |
| d | 80.1 | 93.8 | +13.7 | 52.0 | 41.7 | −10.3 |
| e | 84.4 | 99.2 | +14.8 | 30.4 | 29.5 | − 0.9 |
| f | 60.9 | 90.7 | +29.8 | 48.4 | 35.0 | −13.4 |
| g | 74.9 | 85.8 | +10.9 | 45.6 | 35.4 | −10.2 |
| h | 72.6 | 96.3 | +23.7 | 37.7 | 32.3 | − 5.4 |

| | |
|---|---|
| Mean $pO_2$—before hyperventilation<br>76.4 ± 4.9 | Mean $pCO_2$—before hyperventilation<br>41.1 ± 2.7 |
| Mean $pO_2$—after hyperventilation<br>90.2 ± 5.6 | Mean $pCO_2$—after hyperventilation<br>33.8 ± 1.4 |
| Mean difference—paired comparison method<br>+13.8 ± 3.5 | Mean difference—paired comparison method<br>−7.3 ± 1.6 |
| t = 3.9 P = 0.005 | t = 4.5 P = 0.002 |

*See Table III

(mean increase, 8.6 ± 1.8 mm Hg) and in arterial blood (mean increase, 13.8 ± 3.5). The increases in $pO_2$ in "arterialized" venous blood and in arterial blood were not significantly different (P = 0.21, Table V). Hyperventilation produced a significant decrease in $pCO_2$ in both "arterialized" venous blood and arterial blood. The mean decrease in "arterialized" venous blood was 8.1 ± 2.3 mm Hg, and the mean decrease in arterial blood was 7.3 ± 1.6 mm Hg. The mean decrease in $pCO_2$ produced by hyperventilation in "arterialized" venous blood did not differ significantly from that produced in arterial blood (P = >0.56, Table V). Further analysis of the data shows that the mean $pO_2$ of "arterialized" venous blood and arterial blood before and after hyperventilation showed no significant differences (P = 0.25 and P = 0.112, respectively, Table V). Similarly, the $pCO_2$ levels before and after hyperventilation also revealed no significant differences (P = 0.56 and P = 0.56, respectively, Table V).

TABLE V COMPARISON OF THE EFFECT OF HYPERVENTILATION ON THE $pO_2$ AND $pCO_2$ OF "ARTERIALIZED" VENOUS BLOOD AND ARTERIAL BLOOD—PATIENTS WITH PULMONARY DISEASE

| | "ARTERIALIZED" | ARTERIAL | t | P |
|---|---|---|---|---|
| *$pO_2$, mm Hg* | | | | |
| Mean difference—paired comparison method | +8.6 ± 1.8 | +13.8 ± 3.5 | 1.31 | 0.21 |
| Mean—before hyperventilation | 69.5 ± 3.3 | 76.4 ± 4.9 | 1.16 | 0.25 |
| Mean—after hyperventilation | 78.1 ± 4.6 | 90.2 ± 5.6 | 1.68 | 0.11 |
| *$pCO_2$, mm Hg* | | | | |
| Mean difference—paired comparison method | −8.1 ± 2.3 | 7.3 ± 1.6 | 0.27 | >0.56 |
| Mean—before hyperventilation | 41.2 ± 2.2 | 41.1 ± 2.7 | 0.03 | >0.56 |
| Mean—after hyperventilation | 33.2 ± 2.0 | 33.8 ± 1.4 | 0.25 | >0.56 |

TABLE II. DATA ON THE EFFECT OF HYPERVENTILATION OF $pO_2$ AND $pCO_2$ OF "ARTERIALIZED" VENOUS BLOOD—PATIENTS WITHOUT PULMONARY OR CARDIOVASCULAR DISEASE

| CASE NO. | $pO_2$ MM. Hg |  |  | $pCO_2$ MM. Hg |  |  |
|---|---|---|---|---|---|---|
|  | BEFORE | AFTER | DIFFERENCE | BEFORE | AFTER | DIFFERENCE |
| 1 | 77.9 | 92.5 | +14.6 | 39.3 | 22.7 | –16.6 |
| 2 | 75.7 | 83.4 | + 7.7 | 42.2 | 35.9 | – 6.3 |
| 3 | 72.0 | 78.9 | + 6.9 | 51.4 | 39.1 | –12.3 |
| 4 | 83.9 | 82.4 | – 1.5 | 41.3 | 35.3 | – 6.0 |
| 5 | 69.6 | 92.9 | +23.3 | 39.8 | 28.9 | –10.9 |
| 6 | 69.6 | 78.0 | + 8.4 | 45.8 | 29.7 | –16.1 |
| 7 | 94.0 | 101.4 | + 7.4 | 35.3 | 29.6 | – 5.7 |
| 8 | 83.5 | 97.6 | +14.1 | 35.7 | 32.4 | – 3.3 |
| 9 | 77.5 | 92.6 | +15.1 | 49.7 | 37.7 | –12.0 |
| 10 | 71.9 | 85.9 | +14.0 | 37.3 | 29.8 | – 7.5 |
| 11 | 73.6 | 83.0 | + 9.4 | 47.4 | 36.3 | –11.1 |
| 12 | 78.6 | 87.6 | + 9.0 | 43.8 | 38.9 | – 4.9 |
| 13 | 89.3 | 102.6 | +13.3 | 41.1 | 18.7 | –22.4 |
| 14 | 81.8 | 88.7 | + 6.9 | 43.1 | 38.4 | – 4.7 |
| 15 | 82.2 | 91.0 | + 8.8 | 44.2 | 34.6 | – 9.6 |
| 16 | 72.7 | 85.0 | +12.3 | 42.9 | 31.7 | –11.2 |
| 17 | 83.9 | 90.2 | + 6.3 | 43.8 | 39.2 | – 4.6 |
| 18 | 80.3 | 86.9 | + 6.6 | 44.6 | 34.9 | – 9.7 |
| 19 | 75.2 | 90.9 | +15.7 | 45.6 | 38.5 | – 7.1 |
| 20 | 84.8 | 94.3 | + 9.5 | 39.2 | 26.6 | –12.6 |

| | |
|---|---|
| Mean $pO_2$—before hyperventilation<br>78.9 ± 1.5 | Mean $pCO_2$—before hyperventilation<br>42.7 ± 0.9* |
| Mean $pO_2$—after hyperventilation<br>89.3 ± 1.5 | Mean $pCO_2$—after hyperventilation<br>32.9 ± 1.3 |
| Mean difference—paired comparison method<br>+10.4 ± 1.1 | Mean difference—paired comparison method<br>–9.7 ± 1.1 |
| $t = 9.1$  $P = 0.0001$ | $t = 8.9$  $P = 0.0001$ |

*Standard error of the mean.

TABLE III. DATA ON THE EFFECT OF HYPERVENTILATION ON THE $pO_2$ AND $pCO_2$ OF "ARTERIALIZED" VENOUS BLOOD—PATIENTS WITH PULMONARY DISEASE

| PATIENT | $pO_2$ MM. Hg |  |  | $pCO_2$ MM. Hg |  |  |
|---|---|---|---|---|---|---|
|  | BEFORE | AFTER | DIFFERENCE | BEFORE | AFTER | DIFFERENCE |
| a | 53.0 | 51.8 | – 1.2* | 30.3 | 32.2 | + 1.9 |
| b | 72.4 | 87.6 | +15.2 | 40.9 | 22.3 | –18.6 |
| c | 83.5 | 93.5 | +10.0 | 41.1 | 35.7 | – 5.4 |
| d | 79.3 | 85.5 | + 6.2 | 40.3 | 32.6 | – 7.7 |
| e | 71.2 | 79.0 | + 7.8 | 35.4 | 33.2 | – 2.2 |
| f | 61.2 | 69.2 | + 8.0 | 45.0 | 37.8 | – 7.2 |
| g | 68.8 | 76.6 | + 7.8 | 44.1 | 30.7 | –13.4 |
| h | 66.4 | 81.5 | +15.1 | 52.2 | 40.8 | –11.4 |

| | |
|---|---|
| Mean $pO_2$—before hyperventilation<br>69.5 ± 3.3 | Mean $pCO_2$—before hyperventilation<br>41.2 ± 2.2 |
| Mean $pO_2$—after hyperventilation<br>78.1 ± 4.6 | Mean $pCO_2$—after hyperventilation<br>33.2 ± 2.0 |
| Mean difference—paired comparison method<br>+8.6 ± 1.8 | Mean difference—paired comparison method<br>–8.1 ± 2.3 |
| $t = 4.65$  $P = 0.002$ | $t = 3.56$  $P = 0.007$ |

*Patient *a* appeared to hyperventilate satisfactorily. The results may indicate a diffusion defect due to extensive metastases.

## DISCUSSION AND SUMMARY

This study was concerned primarily with the effect of hyperventilation on the $pO_2$ and the $pCO_2$ of "arterialized" venous blood. A five deep breath hyperventilation method was employed. In the first part of the study, in patients without clinical evidence of pulmonary or cardiovascular disease, it was found that the $pO_2$ was significantly elevated and the $pCO_2$ significantly depressed following hyperventilation.

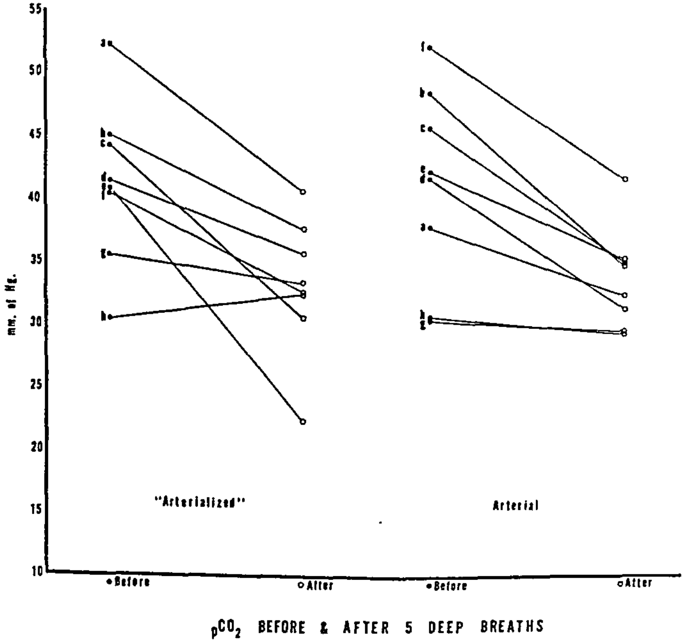


Fig. 4.—The $pCO_2$ of "arterialized" venous blood and arterial blood before and after five deep breaths—8 patients with pulmonary disease.

The second part of the study on patients with pulmonary disease compared "arterialized" venous blood and arterial blood. Comparison of the mean $pO_2$ and $pCO_2$ of "arterialized" venous blood and arterial blood before and after hyperventilation revealed no significant differences. However, using the paired comparison method, it was found that the mean differences between the $pO_2$ of "arterialized" venous blood and arterial blood before and after hyperventilation were statistically significant.

The mean differences in $pO_2$ levels between the two blood samples before hyperventilation were of borderline significance, but after hyperventilation were of definite significance. The mean differences between the $pCO_2$ levels in

In Table VI, the $pO_2$ and $pCO_2$ determinations of "arterialized" venous blood and arterial blood before hyperventilation, and after hyperventilation, were analyzed by the paired comparison method. This analysis revealed a difference of only borderline significance between the $pO_2$ of "arterialized" venous blood and arterial blood before hyperventilation ($P = 0.047$); after hyperventilation, however, the difference between the $pO_2$ levels of the two bloods was significant ($P = 0.001$). The $pCO_2$ levels analyzed by the same method showed no significant differences.

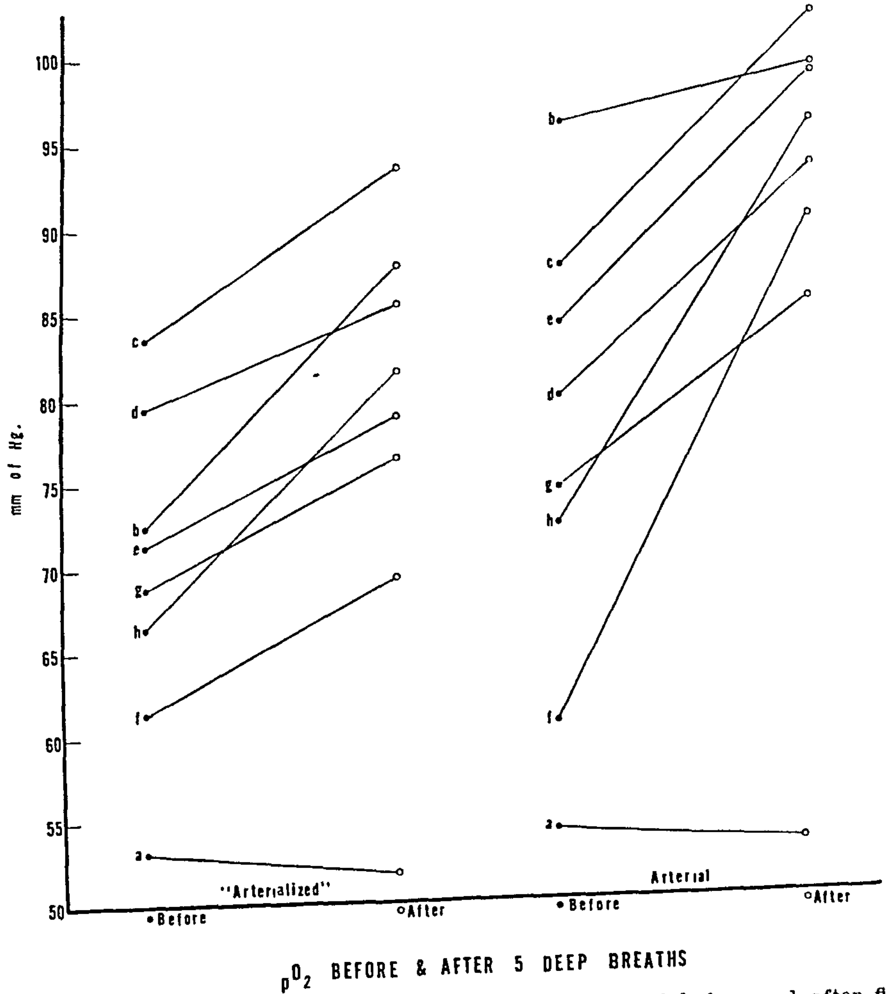


Fig. 3.—The $pO_2$ of "arterialized" venous blood and arterial blood before and after five deep breaths—8 patients with pulmonary disease.

In Part 3, plethysmographic tracings were analyzed by the method depicted in Fig. 5. The amplitude changes and base line shifts were determined. The results of analysis of these studies are shown in Table VII, and graphically presented in Fig. 6. Statistical analysis revealed a significant tendency to vasoconstriction with both types of deep breathing. There were no significant differences in the changes produced by the five deep breath and the 20 seconds of maximal deep breathing. A few patients showed some vasodilatation.

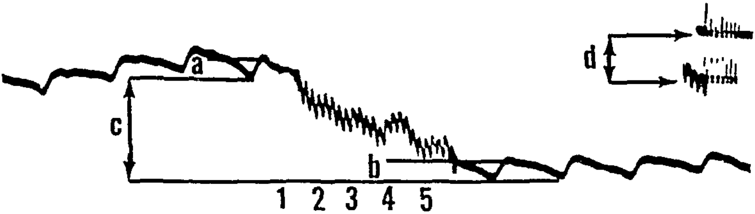
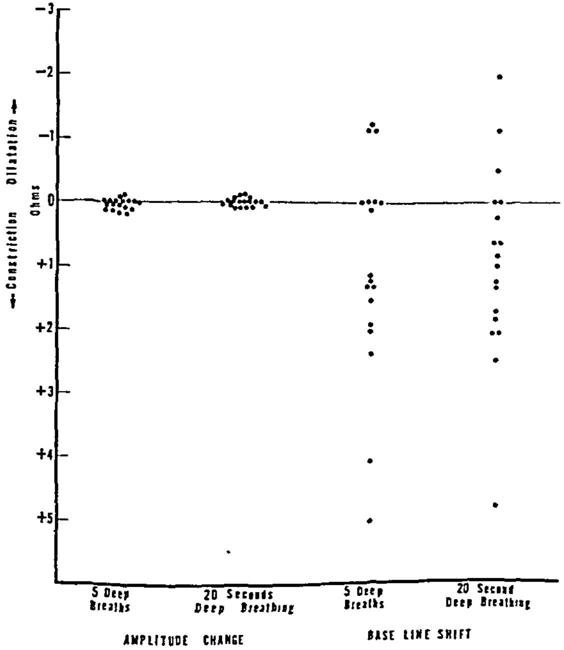


Fig. 5.—Photograph of plethysmographic tracing taken before, during, and after five deep breaths, demonstrating the method of determining the amplitude change and base line shift. The tracing was on slow speed during the five deep breaths *a*, Amplitude before hyperventilation; *b*, amplitude after hyperventilation; *c*, base line shift, and *d*, 1 ohm standard. 1, 2, 3, 4, and 5 indicate the time for each deep breath



CHANGE in FINGER IMPEDANCE

Fig. 6—Scattergram of amplitude change and base line shift—18 patients with no pulmonary or cardiovascular disease—as measured by the electronic plethysmograph before and after five deep breaths and 20 seconds of deep breathing.

TABLE VI. COMPARISON OF THE $pO_2$ AND $pCO_2$ OF "ARTERIALIZED" VENOUS BLOOD AND ARTERIAL BLOOD BEFORE AND AFTER HYPERVENTILATION

| PATIENT | $pO_2$ MM. Hg | | | $pCO_2$ MM. Hg | | |
|---|---|---|---|---|---|---|
| | ARTERIAL | "ARTERIALIZED" | DIFFERENCE | ARTERIAL | "ARTERIALIZED" | DIFFERENCE |
| | | | *Before* | | | |
| a | 54.1 | 53.0 | – 1.1 | 30.6 | 30.3 | – 0.3 |
| b | 96.2 | 72.4 | –23.8 | 42.1 | 40.9 | – 1.2 |
| c | 87.9 | 83.5 | – 4.4 | 41.7 | 41.1 | – 0.6 |
| d | 80.1 | 79.3 | – 0.8 | 52.0 | 40.3 | –11.7 |
| e | 84.4 | 71.2 | –13.2 | 30.4 | 35.4 | + 5.0 |
| f | 60.9 | 61.2 | + 0.3 | 48.4 | 45.0 | – 3.4 |
| g | 74.9 | 68.8 | – 6.1 | 45.6 | 44.1 | – 1.5 |
| h | 72.6 | 66.4 | – 6.2 | 37.7 | 52.2 | +14.5 |
| | Mean difference = –6.9 ± 2.9<br>t = 2.38 P = 0.047 | | | Mean difference = +1.0 ± 2.6<br>t = 0.38 P = >0.57 | | |
| | | | *After* | | | |
| a | 53.2 | 51.8 | – 1.4 | 29.4 | 32.2 | + 2.8 |
| b | 99.6 | 87.6 | –12.0 | 35.5 | 22.3 | –13.2 |
| c | 102.8 | 93.5 | – 9.3 | 31.4 | 35.7 | + 4.3 |
| d | 93.8 | 85.5 | – 8.3 | 41.7 | 32.6 | – 9.1 |
| e | 99.2 | 79.0 | –20.2 | 29.5 | 33.2 | + 3.7 |
| f | 90.7 | 69.2 | –21.5 | 35.0 | 37.8 | + 2.8 |
| g | 85.8 | 76.6 | – 9.2 | 35.4 | 30.7 | – 4.7 |
| h | 96.3 | 81.5 | –14.8 | 32.3 | 40.8 | + 8.5 |
| | Mean difference = –12.1 ± 2.3<br>t = 5.26 P = 0.001 | | | Mean difference = –0.6 ± 2.7<br>t = 0.22 P = >0.57 | | |

TABLE VII. IMPEDANCE CHANGES, INDEX FINGER OF RIGHT HAND, AFTER FIVE DEEP BREATHS AND 20 SECONDS OF MAXIMUM DEEP BREATHING—AMPLITUDE CHANGE AND BASE LINE SHIFT

| NO. CASE | AMPLITUDE CHANGE (OHMS) | | | | | | BASE LINE SHIFT (OHMS) | |
|---|---|---|---|---|---|---|---|---|
| | FIVE DEEP BREATHS | | | 20 SECONDS DEEP BREATHING | | | FIVE DEEP BREATHS | 20 SECONDS DEEP BREATHING |
| | BEFORE | AFTER | DIFFERENCE | BEFORE | AFTER | DIFFERENCE | DIFFERENCE | DIFFERENCE |
| 1 | 0.64 | 0.50 | –0.14 | 0.55 | 0.52 | –0.03 | –2.00 | –0.27 |
| 2 | 0.19 | 0.19 | 0.0 | 0.18 | 0.18 | 0.0 | –2.38 | –1.71 |
| 3 | 0.42 | 0.23 | –0.19 | 0.24 | 0.24 | 0.0 | –1.92 | –1.84 |
| 4 | 0.38 | 0.36 | –0.02 | 0.30 | 0.20 | –0.10 | –1.52 | –2.50 |
| 5 | 0.08 | 0.14 | +0.06 | 0.10 | 0.10 | 0.0 | –1.33 | +0.48 |
| 6 | 0.13 | 0.08 | –0.05 | 0.12 | 0.12 | 0.0 | –0.17 | –0.84 |
| 7 | 0.17 | 0.17 | 0.0 | 0.17 | 0.17 | 0.0 | 0.0 | 0.0 |
| 8 | 0.63 | 0.50 | –0.13 | 0.44 | 0.38 | –0.06 | –4.13 | –2.09 |
| 9 | 0.27 | 0.27 | 0.0 | 0.36 | 0.27 | –0.09 | –1.34 | –2.07 |
| 10 | 0.54 | 0.63 | +0.09 | 0.45 | 0.54 | +0.09 | –1.16 | –1.34 |
| 11 | 0.08 | 0.08 | 0.0 | 0.25 | 0.17 | –0.08 | 0.0 | 0.0 |
| 12 | 0.86 | 0.86 | 0.0 | 0.80 | 0.71 | –0.09 | –5.0 | –0.61 |
| 13 | 0.22 | 0.13 | –0.09 | 0.31 | 0.31 | 0.0 | +1.22 | +1.97 |
| 14 | 0.30 | 0.26 | –0.04 | 0.33 | 0.28 | –0.05 | –1.25 | –1.25 |
| 15 | 0.21 | 0.21 | 0.0 | 0.11 | 0.17 | +0.06 | 0.0 | –0.63 |
| 16 | 0.83 | 0.69 | –0.14 | 0.77 | 0.77 | 0.0 | 0.0 | –4.81 |
| 17 | 0.59 | 0.51 | –0.08 | 0.36 | 0.41 | +0.05 | +1.10 | –1.00 |
| 18 | 0.49 | 0.29 | –0.20 | 0.45 | 0.55 | +0.10 | +1.09 | +1.11 |
| | Mean difference = 0.05 ± 0.02<br>t = 2.7<br>P = 0.015 | | | Mean difference = –0.01 ± 0.01<br>t = 0.8<br>P = 0.43 | | | Mean difference = –1.04 ± 0.40<br>t = 2.61<br>P = 0.02 | Mean difference = –0.97 ± 0.36<br>t = 2.7<br>P = 0.015 |

# In Memoriam

## CARL EGGERS

MARCH 14, 1879—OCTOBER 24, 1956

CARL EGGERS, outstanding example of the best in American surgery came to this country, a fatherless teen age boy, in the 1890's. He brought with him a heavy sense of responsibility to his widowed mother and his younger brother in Oldenburg, Germany. Those close family ties were never broken. Not even the emotional stresses of World War I could affect them. An uncle in Hempstead, Long Island, offered him a home in this new country and a friendly shelter until he could establish himself in the land of opportunity but young Carl had to work his own way. He, also, had to make a very important decision. He wanted, with all his heart, to be a doctor, but that would take time, years of time, it would cost money money that he would have to earn, and, most serious of all, it would postpone the day when he could contribute to his mother's support. There were other professions such as engineering or dentistry, that would yield earlier financial returns but medicine triumphed and it is nice to know that Carl Eggers' mother lived to see her son firmly established in his chosen profession and to receive generously from his earnings. Even so, Carl Eggers had to forego a college education and he was 28 years old when he received his hard won and treasured degree in Medicine from Columbia's College of Physicians and Surgeons in 1907.

What could be more natural than that Carl Eggers should seek for and obtain an internship at New York's "German Hospital" renamed during World War I, the Lenox Hill Hospital. There he served a two and one half year internship, 1908 to 1910 years when foundations were being laid for some of America's contributions to thoracic surgery, Willy Meyer's first lobectomy was performed there in 1909, and the famous "negative pressure chamber" was placed in operation in 1909. From 1910 to 1914 young Dr. Eggers was on duty in the Surgical Dispensary and, in 1914, he became Adjunct Surgeon on the Ward Service. More thoracic history was written at that hospital in those years. Franz Torek carried out the first successful resection of the esophagus in 1913, the bronchoscopist was established as a valued partner of the thoracic surgeon in the care of pulmonary suppuration, and William H. Stewart demonstrated the feasibility of pneumoperitoneum. Carl Eggers, himself often served as anesthetist for Willy Meyer's thoracic operations done in the "negative pressure chamber." Eggers was promoted to Associate Surgeon in 1918, Attending Surgeon in 1924, Consulting Surgeon but such an active participant two years after the usual age limit would compel one to relinquish the title of "arterialized" venous blood and arterial blood before and after hyperventilation showed no significant differences. The comparatively lower $pO_2$ of "arterialized" venous blood after hyperventilation was shown by the impedance plethysmograph to be due to vasoconstriction which occurred in most patients in Part 3 of the study, and reduced to some degree the vasodilation produced by the "arterialization" process.

"Arterialized" venous blood may be used as a substitute for arterial blood in studying changes produced by hyperventilation in the $O_2$ and $CO_2$ tensions. It is felt that the numerical difference in $pO_2$ tensions between the two bloods, as shown by the paired comparsion method, does not preclude the use of "arterialized" venous blood for that purpose.

Statistical methods and data were reviewed in detail by Herman B. Chase, Biometrician and Professor of Biology at Brown University, Providence, R. I.

We wish to acknowledge the technical assistance of Mrs. Dorothy H. Burke, Biochemist, Surgical Research Laboratory, Veterans Administration Hospital, Providence, R. I.

## REFERENCES

1. Weiner, R. S., and Cooper, P.: The Use of "Arterialized" Blood for the Determination of Arterial $O_2$ and $CO_2$ Tensions, J. THORACIC SURG. 30: 683, 1955.
2. Björk, V. O., and Hilty, H. J.: The Change in the Arterial $O_2$ and $CO_2$ Tension During Voluntary Hyperventilation as a Test of Lung Function, J. THORACIC SURG. 27: 541, 1954.
3. Mulinos, M. G., and Shulman, I.: Vasoconstriction in Hand From Deep Inspiration, Am. J. Physiol. 125: 310, 1939.
4. Bolton, B., Carmichael, E. A., and Sturup, G.: Vasoconstriction Following Deep Inspiration, Am. J. Physiol. 86: 83, 1936.
5. Lieb, C. C., Mulinos, M. G., and Taylor, H. L.: Studies on Vasomotor Reflexes. Vasoconstriction From Deep Inspiration of Air, Proc. Soc. Exper. Biol. & Med. 34: 89, 1936.
6. Filley, G., Gay, F. E., and Wright, G. W.: The Accuracy of Direct Determinations of $O_2$ and $CO_2$ Tensions in Human Blood in Vitro, J. Clin. Invest. 33: 510, 1954.
7. Dubois, D., and Nims, L. F.: An Improved Impedance Plethysmograph, Nat. Research Council, Committee on Aviation Medicine, Report, June, 1945.

The Council was therefore empowered to elect, prior to the first annual meeting, such additional members as had already demonstrated their interest and ability in thoracic surgery. Fifteen men were so elected; Carl Eggers was in that group. He was a very active member, rarely missing a meeting, always participating in the scientific discussions, and frequently appearing on the scientific programs as essayist. In 1927, he was elected Treasurer and served for six years; in 1935 he served as Vice President; in 1936 as President; and in 1937 on the Council. Twelve times during his twenty-four years of active membership he served on some committee assignment and after his retirement to senior membership in 1944 he was made chairman of the Association committee to consider the establishment of the Board of Thoracic Surgery. We are all familiar with the very great contribution that he made in that capacity.

In World War I, Carl Eggers applied for and received a commission in the Medical Corps of the Army of the United States. He served as Captain at Camp Jackson and saw the devastating effects of the influenza epidemic and of post-influenzal empyema. A Major, he served as S. G. O. Consultant in Thoracic Surgery. In that capacity, he came to Walter Reed General Hospital in the late summer of 1919. The author of this memoir was on duty there as Chief of the Empyema Section of the Surgical Service. Carl Eggers' mission was to expedite the definitive care of the more refractory cases. He had power, if he felt it advisable, to take over the conduct of the Section. Anything could happen. Courteous, kind, gentle, Carl Eggers became a most welcome guest, welcome alike to personnel on duty and to patients. The Schede operation was the approved method of dealing with chronic empyema cases that had resisted all gentler means. Four "Schedes" were listed each day and the Consultant shared the cases equally with the Walter Reed personnel. What a preceptor he proved to be. The Walter Reed assignment was Carl Eggers' last duty with the Army. Rank was not bestowed lavishly in World War I. Few doctors from civil life received more than a Major's gold leaves. But one day Carl Eggers appeared with the silver leaves of Lieutenant Colonel and the next day he was out of uniform and back in mufti. It was the Army's accepted way of saying "well done" for outstanding professional services.

On March 15, 1919, the day after his fortieth birthday, Carl Eggers married Mrs. Caroline J. Bolter in Chicago, Ill. It was a most happy marriage, as those who have been guests in the Eggers' home can well attest. Mrs. Eggers' father, Edward Henes, had been a trustee of the German Hospital and her brother, Dr. Edwin Henes, was a member of the professional staff. Mrs. Eggers, herself, was vitally interested in the hospital's welfare and worked hard on the Ladies Aid Society. Mrs. Eggers, her two sons, and five grandchildren survive Carl Eggers.

For all his heavy duties at Lenox Hill Hospital, Carl Eggers found time to serve on the surgical staffs of New York Skin and Cancer Hospital (Associate, Attending, Director of Surgery), as Director of Surgery at Stuyvesant "attending." During those active years Carl Eggers contributed his share to the growth and expansion of thoracic surgery. He is credited with the fourth successful resection of the esophagus for carcinoma, in this country.

His whole professional life was closely identified with the Lenox Hill Hospital. His office was just across the street; he lived just across the street; to that hospital went his library, his office equipment, and his record files and, in return, out of love and affection for the man who had served so long, so generously, and so selflessly, the Trustees of Lenox Hill Hospital voted to name the operating room suite of the planned new building, "The Dr. Carl Eggers Operating Suite." Word of that honor came to Carl Eggers during his last sickness. He was deeply moved. The subscription books have not yet been closed on the funds for that new building program.

CARL EGGERS
1879-1956

Carl Eggers was elected to membership in the American Association for Thoracic Surgery in the interval between the organization meeting of 1917 and the first Annual Meeting, in 1918. At the time of the "luncheon meeting at the Waldorf" in June, 1917, when the Association was organized, it was realized that many men who should have been included with the fifty founders were prevented from attending by reason of wartime duties or circumstances.

The Journal of
Thoracic Surgery

VOL. 33 APRIL, 1957 No. 4

## Original Communications

# MITRAL COMMISSUROTOMY PERFORMED FROM THE RIGHT SIDE

CHARLES P. BAILEY, M.D.,* AND DRYDEN P. MORSE, M.D.**
PHILADELPHIA, PA.

### HISTORICAL ASPECTS OF MITRAL COMMISSUROTOMY

THE development of an efficient surgical treatment for mitral stenosis has, like most other significant medical advances, been preceded by a long period of observation and investigation, formulation, and finally clarification of concepts. After an interval of trial and error, significant clinical success has been achieved in a large number of cases. However, there has been no cessation in our efforts to extend the benefit of surgery to a wider group of patients and also qualitatively to improve the individual result. The present report describes a new development which, it is felt, has much to offer. The historical background of this conceptional change is interesting and pertinent.

The first good description of the pathologic entity which we know as mitral stenosis was given by de Vieussens[1] in 1705. In 1819, Laennec[2] correlated the clinical features of the disease with the observed pathologic changes, discussed the probable disturbances of physiology, and described the auscultatory findings which make an accurate diagnosis possible during life. A direct surgical attack upon the stenotic valve in order to enlarge the orifice ("slightly notching the valve") was proposed by Samways,[3] in 1898, and by Brunton,[4] in 1902. The latter specified that the surgical incisions should lengthen the valve slit.

---

This work done during tenure of a National Heart Institute Fellowship.

This work was supported by the Mary Bailey Foundation for Heart and Great Vessel Research, Philadelphia, Pa.

All of the catheterization and other physiologic data were supplied by Dr. Harry Goldberg and The Brith Sholom Cardiopulmonary Laboratory at Hahnemann Medical College and Hospital, Philadelphia, Pa.

Received for publication March 21, 1956.

*Professor and Head of Department of Thoracic Surgery, Hahnemann Medical College and Hospital, and Director, Bailey Thoracic Clinic, Philadelphia, Pa.

**Resident, Department of Thoracic Surgery, Hahnemann Medical College and Ho[illegible] Philadelphia, Pa.

Square Hospital, to serve as Clinical Professor of Surgery on both the Columbia and the New York University Medical Faculties and as Consultant Surgeon to three other New York City hospitals.

He was particularly interested in the training of physicians, young and old. For six years he was chairman of the Committee on Medical Education of the New York Academy of Medicine. He was credited with outstanding service in this field.

To the young men who passed through Lenox Hill Hospital as intern or resident, he endeared himself. Herbert C. Maier has spoken well for this group:

> Those young surgeons who had the good fortune to receive training under the guidance of Doctor Carl Eggers well realize that he exemplified the true physician. By his daily acts in the intimate personal care of his patients he demonstrated to his associates and house staff that in careful and sympathetic attention to the surgical, medical and personal problems of the patient lay the path to the highest type of medical care. Though a busy surgeon, he did not delegate wearisome details to others but obviously regarded them as his responsibility so that the patient could place full trust in him. Little wonder that such confidence was engendered in his patients.
>
> Doctor Eggers also exemplified that an attending on the staff of a hospital not closely affiliated with a medical school may nevertheless be at the forefront of surgical teaching and contribute to scientific advances. Doctor Eggers had a profound interest in the personalized training of young surgeons and he had the foresight to recognize the pitfalls of early narrow specialization. He therefore urged that a broad training in general surgery be obtained irrespective of the degree of specialization later to be practiced.

Honors came to Carl Eggers in abundance. A few days after his seventy-fifth birthday (1954), he was guest of honor at a surprise dinner given in his honor by the executive, administrative, and professional staffs of the Lenox Hill Hospital in the Waldorf-Astoria Ball Room. He was the recipient of a gold medal commemorating the occasion and of an album of pictures taken of those in attendance. Later, in 1954, he paid a visit to his birthplace, Oldenburg, Germany, and was made an honorary citizen of the town. Carl Eggers had to sign the roll book of honorary citizens and enter some short remark. Mrs. Eggers reported that he thought a moment, then smiled, and in fine German script wrote briefly and simply his life's philosophy: *It is so easy to do good in the world and it makes one so happy.*

—ETHAN FLAGG BUTLER, M.D.

Harken's[10] original "valvuloplasty" procedure. On June 16, 1948, he successfully enlarged a stenotic mitral valve orifice by instrumental resection of portions of the mural valve leaflet at the price of what he called "selective insufficiency."

TECHNICAL FEATURES OF MITRAL COMMISSUROTOMY AS PERFORMED FROM THE LEFT SIDE.—From that point, up to the present time, there have been relatively few major changes in the over-all method of commissural separation. The right index finger is passed into the left atrium through an incision made in the tip of the left auricular appendage. Resistant valves are separated at the commissures by instrumental means, while the more amenable are split by manipulations of the intracardiac finger alone. Whenever the subvalvular structures are cross-fused below the valve orifice, as described by Brock,[11] they must be split apart or divided instrumentally as described by Nichols[12] One major advance has been the introduction of the "bare finger" technique by Husfeldt.[13] This increases the operator's tactile perception and hence accuracy of manipulation by several hundred per cent.

In the majority of the patients who have been operated on up to the present time, only the anterolateral commissure has been separated. The effective reason for this is as follows: The posteromedial commissure seemingly is appreciably more resistive to blunt disruptive pressure as it is applied from the left side, and instrumental attempts at division of this portion of the valve substance (from the left) often are attended by the creation of considerable valvular incompetence.

In approximately 20 per cent of the first 1,200 patients operated on at our Clinic, there was recognized, in addition to the obliteration of the commissures, a significant degree of subvalvular obstruction to blood flow due to a complex interfusion or cross-fusion of the chordopapillary supporting tissues This actual hindrance to blood flow must be distinguished from clinically insignificant instances of thickening and fusion of contiguous chordae which may be demonstrated to exist in the vast majority of patients with clinical mitral stenosis In most instances, any secondary obstruction has been overcome readily by direct digital pressure. In some patients, in whom the papillary muscles are directly adherent to the valve apex, instrumental splitting of these muscles, as described by Nichols,[12] may be necessary in order to establish an adequate subvalvular passageway. This was carried out in six instances in the present series. Such mechanical separation, whenever necessary, of subvalvular "adhesions" of the chordopapillary system to permit free separation of the leaflets logically is included within the term "commissurotomy" in the same sense that separation of pleural adhesions and mediastinal dissection, when necessary, are included within the term "pneumonectomy."

Good clinical results have been reported upon sizable series of operative patients by numerous authors.[11, 14-16] However, the periods of follow-up have been relatively short in the over-all course of rheumatic valvular disease and the reported "long-term" benefits have been limited approximately to 65 to 75 per cent of those so treated (Table I).

Cutler's[5] pioneering intervention, in 1923, using a valvulotome and the transventricular route in which he converted a stenotic mitral valve aperture into a larger, although less competent one, set the pattern of surgical attempts for the next twenty-odd years, culminating in the brilliant efforts of Smithy[6] who performed Cutler's procedure on 7 patients with five survivals. However, the considerable amount of regurgitation necessarily produced by these attempts militated against true clinical success and often precluded operative survival.

Souttar,[7] in 1925, on the other hand, successfully "dilated" a stenosed mitral valve by manipulations of the index finger which had been inserted into the left atrial chamber through an incision made in the tip of the left auricular appendage.

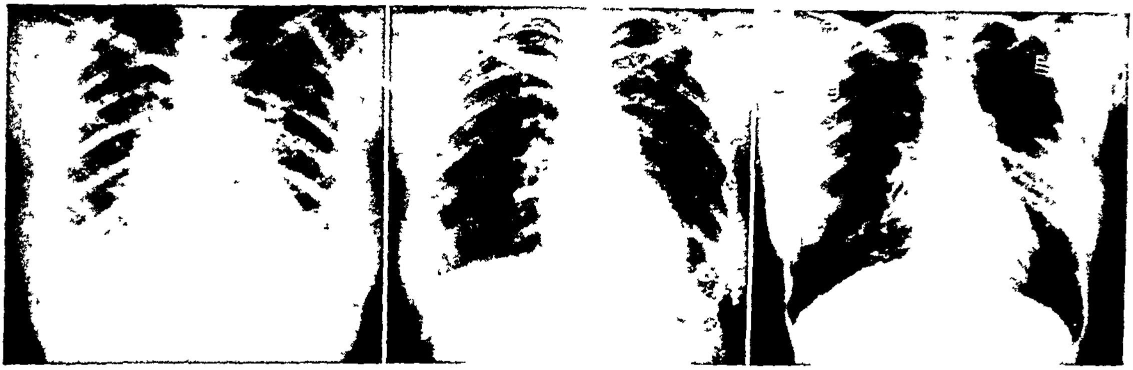


Fig. 1.—Preoperative, six months postoperative, and 6½ years postoperative (left to right), follow-up roentgenograms in C. W., the first patient historically to undergo a modern type of operation for mitral stenosis. (Courtesy Pennsylvania M J. 53: 6, 1950.)

In 1948, after preliminary unsuccessful clinical attempts dating back to 1945, the senior author presented the concept of separating the fused valve leaflets anatomically along the lines of the obliterated commissures, thus overcoming the valvular obstruction without the inherent necessity of producing any regurgitation. This concept is the basis of the modern corrective operation for mitral stenosis, and has been designated as "commissurotomy."[8] The first successful intervention of this type in a patient with classical mitral stenosis was accomplished on June 10, 1948. A hooked knife attached to the finger was used to divide the valve. The mobilized leaflets, thereby, were rendered capable of function of a normal type; no regurgitation was created. The auscultatory evidences of disease have been nearly abolished, and the heart size has become reduced progressively since operation (Fig. 1). This young woman continues to remain in excellent health to the present time more than eight years after operation.

Independently of the American investigations, in September, 1948, Brock[9] successfully carried out an operative procedure which was based upon a similar concept of mobilizing the fused leaflets, but which was accomplished by splitting with finger pressure alone. A logical intermediate step, although chronologically performed later, between those procedures in which regurgitation necessarily was produced and the modern type of operation, was

TABLE I RESULTS IN FIRST 1,000 PATIENTS OPERATED UPON FOR MITRAL STENOSIS—LEFT SIDED APPROACH (LONGEST FOLLOW UP—8 YEARS)—AUTHOR'S SERIES

| | NUMBER OF CASES | PERCENTAGE |
|---|---|---|
| Mitral commissurotomy | 1,000 | 100 |
| Operative deaths (excluding 2 cases of suicide) | 77 | 7.7 |
| Late deaths (2 of these deaths were due to carcinoma) | 56 | 5.8 |
| Follow up of survivors | | |
| Improved | 711 | 89.8 |
| Unchanged | 49 | 6.2 |
| Worse | 32 | 4.0 |

Unable to follow up 73 cases

ANALYSIS OF UNSATISFACTORY RESULTS.—Leaving out of consideration for the moment those patients who do not survive operation (2 to 10 per cent), we find an appreciable number (10 to 15 per cent) who are not helped initially by the procedure, or are even made worse by it. This group includes patients in whom the stenosis has been relieved but slightly or not at all, and also those in whom a significant amount of regurgitation has been produced. It does not include those patients who suffer postoperatively from recognized or unrecognized pleural or pericardial effusion which ultimately resorbs with clinical improvement. A presently indeterminate number of patients exhibit definite and often marked improvement for a variable number of months or years but gradually redevelop symptoms and signs similar to those which brought them to the cardiac surgeon initially. In such individuals, left heart catheterization nearly always reveals definite evidence of re-establishment of an obstruction to blood flow at the level of the mitral valve—elevation of diastolic gradient between left *atrium and ventricle* (Fig. 2).

A frequently unsuspected but relatively common cause of an unsatisfactory clinical result is unrecognized or incompletely appreciated coexistent disease of another valve—either the aortic or tricuspid. That such rheumatic multivalvular involvement is not uncommon is clearly shown in Table II. It will be noted in this table that in the clinical studies tricuspid disease was recognized rarely, if at all, while its incidence in the autopsy studies ranged from 10 to 25 per cent.

*Recurrent Mitral Stenosis.*—Brock,[17] Jordan and Hellems,[18] Keyes and Lam,[19] and others have reported cases in which they have attributed clinical regression to restenosis of the valve. In some, the diagnosis has been confirmed by reoperation and direct palpation of the valve. In others, the opinion has been based upon subsequent autopsy findings. It is our belief that, with the increasing number of mitral interventions and the passage of time, such reports will become more frequent in the near future.

Harken and Black,[20] and Glover and his associates[21] have objected strongly to the concept of recurrence of mitral stenosis, believing restenosis of the surgically well opened valve to be extremely improbable. They feel that such instances of re-establishment of obstruction at the mitral level are due basically either to incomplete opening of the fused commissures at the original operation or to failure

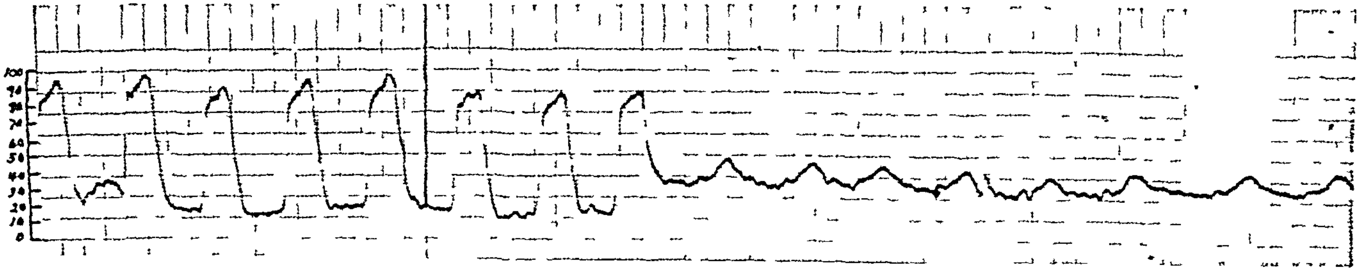


Fig. 2—Pressure tracing *obtained* in R. H. during left heart catheterization as catheter was withdrawn from the left ventricle into the left atrium The gradient of 30 mm Hg existing across the mitral valve during left ventricular filling indicated a severe state of restenosis of the mitral valve in a woman who had been markedly improved for the first three of four years following mitral commissurotomy Reoperation from the right side confirmed the diagnosis (8 mm by 3 mm valve slit) and overcame the obstruction much more adequately than had been accomplished originally (from the left) (Courtesy H. Goldberg, Brith Sholom Cardiopulmonary Laboratory, Philadelphia Pa )

reoperated on 2 patients in whom complete bipolar commissurotomy had been accomplished previously and in whom extensive refusion of the commissures had taken place. These are being reported in detail elsewhere. Probably there have been many unrecognized cases of this condition among the great number of patients who have been subjected to mitral valve surgery throughout the various clinics in the world, since the possibility of its occurrence has not been widely appreciated. Doubtless some of these individuals, even now, erroneously are presumed to be suffering from irreversible myocardial or pulmonary vascular disease.

The extreme pathologic changes which have taken place in many of the valves of our operative patients must tend to reproduce or to perpetuate the tendency of the process to produce complete obliteration of the orifice. Slowly moving, stiffened valves, even though divided well beyond the scarred margin of the passageway, may remain "hinged" upon partially fibrosed tissue. Further, it would seem that the surgically produced irregularities of the cut valve surfaces, as well as those produced by the existing pathologic alterations, must continue to produce the flow turbulence to which Magarey[23] attributes the recurrent surface deposition of fibrinous exudate which appears to be the basic element in the pathogenesis of mitral stenosis. In all probability, once the valve pathology has become advanced sufficiently, the stenosing process, even though set back mechanically by one or more successful surgical interventions, inevitably must continue to progress as long as life exists. In this respect, a very severe stenotic deformation of the mitral valve apparently behaves like the fibrous stricture which it really is.

On the other hand, a mitral obstruction, which may be just as tight mechanically as the preceding type, may be found unassociated with extreme pathologic change in the leaflets. Extensive but light cross-adherence of the leaflets' margins has greatly reduced the size of the valve aperture. It would appear improbable that these flexible and mobile cusps would become re-fused extensively following adequate surgical separation unless the patient were to suffer from another bout of rheumatic fever.

While in valves with the extremes of pathology, recurrence of mitral stenosis would seem to be inevitable, regardless of the surgical technique employed, and while in others with minimally diseased leaflets, re-fusion following separation would seem to be extremely unlikely, there is a large intermediate group of patients. In them, it is probable that the adequacy of surgical valve mobilization largely determines the likelihood and rate of reestablishment of the obstruction at the mitral level.

Certainly it would seem logical that, following a previous commissural separation, the unquestionably rheumatic patient would be likely to redevelop a stenotic valvular lesion in the event that he should suffer another bout of rheumatic "activity." And, in any case, whether due to specific exacerbation or to residual mechanical irregularities of the valve surfaces, it seems reasonable to assume that this admitted tendency toward restenosis must be greater with respect to the valve leaflets than to the subvalvular supporting structure

TABLE II. INCIDENCE OF VALVULAR HEART DISEASE*

| | NEW ENGLAND | | VIENNA | BERLIN | JOHNS HOPKINS, BALTIMORE |
|---|---|---|---|---|---|
| | CABOT | WHITE | KAUFMANN | SPERLING | HIRSCHFELDER |
| | 208 CASES POST-MORTEM (%) | 1,097 CASES CLINICAL (%) | 126 CASES POST-MORTEM (%) | 300 CASES POST-MORTEM (%) | 1,781 CASES CLINICAL (%) |
| Mitral | 85.6 | 56.3 | 60 | 85 | 51 |
| Aortic | 44.7 | 14.7 | 76 | 43 | 22 |
| Tricuspid | 15.9 | | 25 | 10 | |
| Pulmonary | 1.9 | | | 1 | |
| Aortic and mitral | 19.2 | 28.9 | | | 20 |
| Aortic, mitral, and tricuspid | 11 | | | | |
| Mitral and tricuspid | 2.9 | | | | |
| Pulmonary and tricuspid | 1 | | | | |
| All four valves | 1 | | | | |

*From Paul D. White, M.D., Heart Disease, ed. 4, New York, The Macmillan Publishing Company.

of the operator to relieve a subvalvular obstruction. We agree that such incomplete surgery predisposes to early secondary narrowing of the valve passageway and must be a major factor in the re-establishment of mitral obstruction. However, this very re-establishment of the stenosis after initial partial relief must, by definition, be admitted to constitute *recurrence* of the obstruction.

Indeed, we feel the precept, which one of us (C. P. B.) formerly advanced,[8] that wide opening of but a single commissure constitutes an adequate surgical mobilization of the valve in most cases, has done much harm. Similarly, Harken's[10] strongly promulgated dictum (which he has since abandoned[22]) that an adequate splitting of the valve may be accomplished nearly always by digital pressure alone (instrumentation, therefore, rarely being necessary), has been equally unfortunate. It is now clearly evident to both proponents of these erroneous concepts that the widest feasible mobilization of both obliterated commissures is indicated and that an instrumental attack is necessary to accomplish this in at least one-half of all operative patients. Undoubtedly, these early misconceptions have vitiated, in certain respects, the value of the statistical follow-up upon both these sizable operative series. Furthermore, these precepts, having been accepted by many other surgeons as sound, have redounded to the disadvantage of their patients as well as themselves. No doubt many patients so treated will become candidates ultimately for more adequate reoperation.

*Recurrent Stenosis After Complete Mobilization of the Valve.*—There remains a group of patients with indubitable recurrent stenosis of the adequately opened and mobilized valve. The occurrence of this condition can be established only if a surgeon of considerable experience in this field carefully reports the initial operative result and then, months or years later, personally reoperates on this patient or observes the valve at autopsy.

We are able to report fourteen instances of proved recurrent stenosis in patients who had had unicommissural mobilization of the valve. We also have "digital vision" Often, full valvular mobilization is extremely difficult to accomplish Further, the operation inherently is associated with dangers of causation of cardiac arrest, arterial embolization, and mitral regurgitation To permit even a naturally skillful and generally well trained surgeon who happens to be unfamiliar with this technique to attempt such an operation, which must be performed by the guidance of the sense of touch alone, is a reflection upon our medical intelligence and integrity If it takes the accepted minimum of 50 transurethral resections, properly performed under supervision, to prepare a novitiate for the practice of that phase of the specialty of urology,"[6] it would seem that at least an equal number of episodes of training must constitute the minimum preparation acceptable for the practice of the surgical treatment of mitral stenosis

DISSATISFACTION WITH MITRAL COMMISSUROTOMY OPERATIONS AS USUALLY PRACTICED —While the operation of mitral commissurotomy performed from the left side has been becoming gradually more acceptable to the profession and even has achieved high favor in some circles, the shortcomings of the standard technique have become increasingly obvious to your authors For a long time, no satisfactory solution to these problems has seemed to be within reach Difficulties experienced in simultaneously treating coexisting rheumatic lesions of the aortic and tricuspid valves and the irreducible incidence of arterial embolization occurring during mitral surgery have increased this dissatisfaction, although our major complaint has been related to the unsatisfactory technical accomplishments

### THE RIGHT SIDED APPROACH

In a letter to Sir Horace Mann, dated Jan 28, 1754, Horace Walpole[7] said that he invented the term "Serendipidy" on the title of a fairy tale "The Three Princes of Serendip," because the princes were "always making discoveries by accident and the sagacity of things they were not in quest of" In the same sense the discovery of the advantages of performing mitral commissurotomy from the right side surely must be accredited to Serendipidy since it was incidental to attempts to perform simultaneous surgery for right sided noncardiac lesions

In 1954, the first description of an approach to the mitral valve from the right side, with a report upon 3 clinical patients was presented by Neptune and Bailey[8] At that time it was felt that the indications for such an approach in mitral stenosis were relatively few, consisting mainly of the coexistence of right sided extracardiac lesions amenable to surgery or of definitely diagnosed multivalvular stenosis (aortic or tricuspid) which would require a right thoracic intervention Since the submission of that report, further advances in the development of the technique and a better understanding of the utility of this approach have caused us gradually to adopt it as our routine method for the performance of mitral commissurotomy in the absence of appropriate contraindications Some of the advantages and disadvantages of the right sided operation are listed in Table III

This order of likelihood would seem to be implicit in the designation of such subvalvular changes as "secondary stenosis," the actual leaflet lesions causing "primary stenosis."

Whatever the actual incidence of recurrent stenosis following adequate commissurotomy may prove to be eventually, it is probable that most instances of re-establishment of obstruction at the mitral valve up to the present time have been favored by incomplete surgery, commissural separation having been extended insufficiently or an element of coexisting subvalvular stenosis having been overlooked or ignored. All experienced cardiac surgeons, in retrospect, can recall instances in which they have failed in these respects.

INADEQUATE SURGICAL EXPERIENCE.—Tragically, as yet, there appears to be little organized effort on the part of the profession to prevent further errors of this type from being made by beginning cardiac surgeons in the future. A young and enthusiastic surgeon with grossly inadequate training in the field of cardiac surgery may not hesitate to insert an index finger into the left atrium with the intention of performing mitral commissurotomy, even though, as an extreme example, he may never have performed any major cardiovascular operation previously. Repeatedly, the senior author (C. P. B.) has been astonished to meet surgeons who never have obliterated a patent ductus arteriosus or resected a coarctation of the aorta, but who claim to have done a number of "mitrals." It is extremely unlikely that they have completely split the valves in 50 per cent of the cases which may be reasonably amenable to digital separation of the commissures. Certainly they have not effectively treated the 50 per cent which require instrumentation because they have used no instrument. More likely, each has swept a finger around the atrial chamber in a state of complete disorientation and then has removed the digit triumphantly from the heart without even palpating the valve orifice. Since the finger and valve cannot be seen, who is to know what really has transpired within the heart? Usually, with modern anesthesiologic and cardiologic control, the patient will survive and the case will be listed, again triumphantly, as a successful mitral commissurotomy operation. With the intense psychologic impression which such an operation creates, and with the interested and more concentrated medical attention which then is accorded him, the patient may feel much improved during the next few months. Ultimately, as real improvement fails to materialize and the disease progresses sufficiently, he will deteriorate perhaps to be listed as a case of "recurrent stenosis." This tragic situation is reminiscent of that which persisted for some time after the introduction of the endoscopic transurethral method of prostatic resection.

TECHNICAL DIFFICULTY OF THE OPERATION.—Such amateurish surgical attempts already have brought about a considerable amount of discredit to the operation of commissurotomy.[24, 25] Yet, experience and ordinary common sense indicate that this operation is a highly technical one, frequently requiring skillful use of special instruments under the sometimes unfamiliar guidance of

TABLE III, A. ADVANTAGES OF THE RIGHT SIDED APPROACH

1. A superior mitral commissurotomy may be accomplished with full and safe mobilization of both commissures
2. Unsuspected and marked coexistent tricuspid disease may be diagnosed readily and treated appropriately
3. Coexistent aortic stenosis may be relieved by the preferred transaortic technique through the same operative incision
4. Operative arterial embolization due to manipulative dislodgment of thrombotic material from the atrial appendage may be avoided
5. The supine position is physiologically easier on the patient
6. A coexistent unsuspected interatrial septal defect may be diagnosed digitally and treated at the same operative session
7. Concomitant right sided noncardiac disease may be treated simultaneously
8. Conditions involving the left thorax may render left thoracic cardiotomy more difficult or dangerous, this refers particularly to previous left sided mitral intervention, also thrombotic obliteration of the appendage, and the rare condition of calcification of the left atrial wall
9. The incision is shorter and less traumatic, and causes less postoperative pain and shoulder dysfunction
10. The skin incision averages 27 cm. in length compared to 47 cm with the left posterolateral incision, and is hidden largely within the inframammary fold in the female patient (cosmetic advantage)

TABLE III, B. DISADVANTAGES OF THE RIGHT SIDED APPROACH

1. The pertinent anatomic orientation is entirely different from that of the left sided approach, and must be learned before the commissures can be mobilized accurately and fully
2. For the surgeon inexperienced in this approach, hemostasis is technically more difficult to achieve because there is no appendage upon the right side of the left atrium
3. Pressure on the right side of the heart may produce severe although transient hypotension
4. It is more difficult to extract a loose thrombus from the left atrium from the right side
5. The left auricular appendage cannot be amputated in an effort to prevent recurrent arterial embolization; however, this amputation is believed now to be unnecessary with the relief of stasis afforded by satisfactory enlargement of the valve
6. Cardiac massage, if necessary, cannot be carried out effectively from the right side in most instances
7. Should an unexpectedly large amount of mitral insufficiency be encountered, the appropriate corrective measures cannot be applied from the right side (see Fig 6, *A*, *B*, *C*)

An objection has been raised to the performance of mitral commissurotomy through a right-sided incision on the basis that it is "an operation for a left-sided lesion performed from the right." Yet, this same critic[29] very recently has recommended the performance of tricuspid commissurotomy (truly a right-sided lesion) through a left thoracic approach!!

In reality, as shown in Fig. 3, the left atrium extends anatomically just as far toward the right side as toward the left It more properly might be designated at the posterior atrium. Actually, the mitral valve orifice does lie slightly to the left of the midline, much as the entire heart is directed somewhat to the left (Fig 4). However, this minor anatomic deflection is of slight importance in comparison with the many technical and pathologic features which favor the right-sided route for mitral commissurotomy.

This report is based upon our experience with 200 patients who were operated on by the right-sided approach for significant mitral stenosis, with

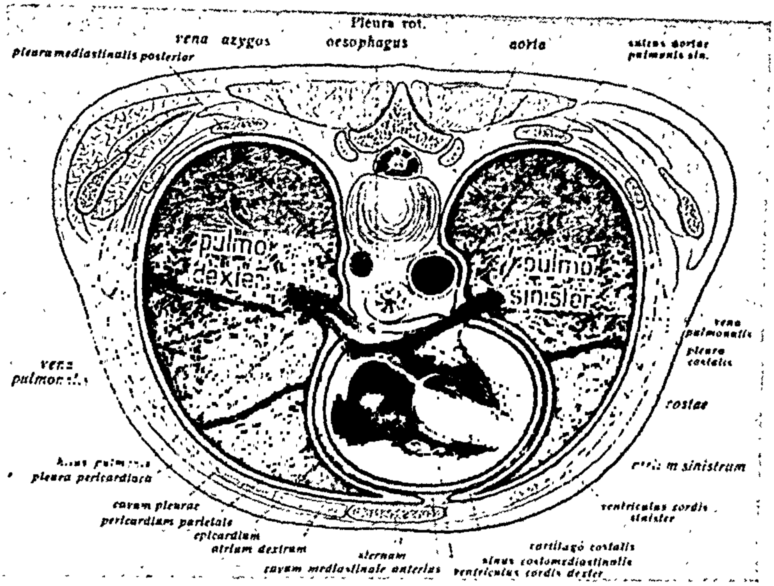


Fig. 3.—Diagrammatic representation of relationships of cardiac structures at the level of the mitral valve. Note that the left atrial chamber extends fully as far toward the right side as toward the left, and that it logically might be called the "posterior" atrium. (Courtesy Sobatta, München, 1920, J. F. Lehmanns Verlag.)

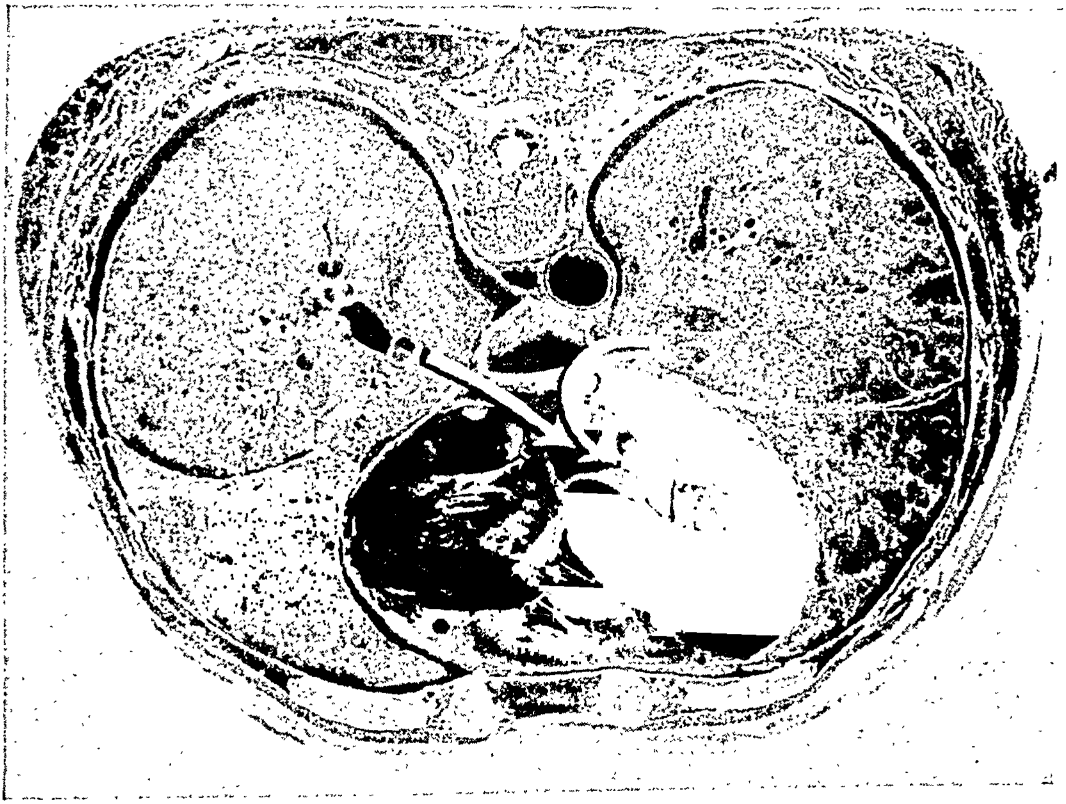


Fig. 4.—Actual cross section of human thorax at the level of the mitral valve. Note that while the mitral valve orifice actually does lie somewhat to the left of the midline, the approach to it by way of the left auricular appendage (see arrow) is basically an awkward one in that the finger after insertion must practically be doubled back upon itself to reach the commissures. In contrast (see arrow), the right-sided approach brings the finger tip to the valve in direct end-on fashion which greatly facilitates all manipulations. (Courtesy P. Seara, Estudio Anatomico Topografico Del Torax y Del Abdomen, Buenos Aires, Argentina, 1941).

The above statistics are compared with our experience in the first, 1,200 patients with mitral stenosis upon whom we operated from the left side (Table V). In them, bicommissural separation was accomplished in 33.1 per cent. The anterolateral commissure alone was separated in 65 per cent. The posteromedial alone was divided in 1.9 per cent. It is our considered opinion that the average size of the opening achieved when the operation is performed from the right side ranges from 33 per cent to 300 per cent larger than that which may be expected with the left sided approach (Table VI). This additional enlargement of the orifice is not attended by any increase in the incidence of significant mitral regurgitation which might be created or magnified by the surgery.

TABLE V. NUMBER OF COMMISSURES OPENED FROM THE RIGHT SIDED APPROACH COMPARED WITH THE LEFT SIDED APPROACH

| | LEFT SIDED APPROACH (1,200 CASES) (%) | RIGHT SIDED APPROACH (196 CASES*) (%) |
|---|---|---|
| Both commissures opened | 33.1 | 97.3 |
| One commissure only | | |
| Anterior | 65.0 | 1.6 |
| Posterior | 1.9 | 1.1 |
| Total | 100 | 100 |

*Four cases operative data not complete.

TABLE VI. VALVE SIZE ATTAINED IN MITRAL COMMISSUROTOMY FROM THE RIGHT SIDE AS COMPARED WITH THE RESULTS FROM THE LEFT

| FINAL VALVE SIZE | RIGHT SIDE | | LEFT SIDE | |
|---|---|---|---|---|
| | NUMBER OF CASES | PER CENT | NUMBER OF CASES | PER CENT |
| Less than 1½ finger opening (up to 2.4 $\text{cm}^2$) | 5* | 2.5 | 23 | - |
| 1½ finger opening (2.5 $\text{cm}^2$) | 56 | 28.0 | 549 | 46 |
| 2 finger opening (4 $\text{cm}^2$) | 133 | 66.5 | 519 | 43 |
| Over 2 finger opening (4 cm. plus) | 6 | 3.0 | 30 | 2 |
| Unknown | 0 | 0 | 79 | 7 |
| Total | 200 | 100 | 1200 | 100 |

*Three of these were table deaths with the valve surgery not completed.

*Subvalvular stenosis.* We have observed no reduction in the facility with which subvalvular chordopapillary fusion masses may be divided. If unseparated, they may prevent otherwise adequately mobilized valve leaflets from separating during diastole. If they are sufficiently extensive, they may provide a secondary site of atrioventricular obstruction below the level of the actual valve orifice. In 29 of the 200 patients with mitral stenosis operated upon from the right side, thickening and cross fusion of the chordopapillary supports was recognized and relieved, in 6 by definitive instrumentation. Although *in most instances such secondary obstruction can be overcome* readily by direct digital pressure, there are some patients in whom the papillary muscles may be directly adherent to the valve apex and instrumental splitting of these muscles, as described by Nichols,[12] may be necessary in order to establish an adequate subvalvular passageway.

or without coexistent aortic or tricuspid disease. The first patient was operated upon on Nov. 1, 1954, and the last on Oct. 8, 1955.

ADVANTAGES OF THE RIGHT-SIDED APPROACH.—

*An Improved Commissurotomy.*—While originally the performance of commissurotomy from the right side was described[28] as technically more difficult than that through the usual left auricular appendage approach, and while, at first, our various staff surgeons* felt insecure and less than normally competent while attempting to separate the fused valve commissures from this new and "unusual" approach, subsequent experience has brought about improved orientation and greatly has increased our technical skill in the various manipulations entailed in the performance of commissurotomy from the right side. Today, we are of the unanimous opinion that a markedly superior mobilization of valvular structures may be achieved from the right than usually is feasible from the left. The term "mobilization" is used advisedly. By it we mean, not only, the anatomic separation or splitting apart of the fused leaflets, but also, the restoration of leaflet function by separating any cross-fused subvalvular supports.

In contrast to the general experience that it is difficult to open the posteromedial commissure from the left side, when the mitral valve is approached from the right, this commissure lies conveniently in line with and *just below the site of cardiac* entrance and may be opened accurately and safely by digital or instrumental technique in nearly every instance. This valve extremity has been opened in all but 3 of our first 200 patients operated on from the right side. Such separation of the posteromedial commissure rarely is attended by the creation of significant regurgitation. It is somewhat more difficult to separate the anterolateral valve commissure by digital pressure alone from the right side, partly because of the difficulty in applying external counterpressure. However, instrumental division is found to be no more difficult than when using the left lateral approach and either instrumental or digital opening of the fused anterolateral commissure was also accomplished in all but 2 of the 200 cases. Table IV shows the reason why the other commissure was not opened in the 5 cases in this series in which only one was attacked. The directions of approach to the valve orifice from the respective two sides are contrasted in Fig. 4.

TABLE IV. THE REASON FOR NOT OPENING A COMMISSURE IN THE 5 CASES WITH ONLY ONE COMMISSURE OPENED

H. M.—loosely attached calcium in posteromedial commissure—feared brain embolus. Anterolateral opened

E. B.—*eccentric orifice* extending anteriorly to the valve ring; posteromedial commissure opened completely

C. H.—chordae tendineae adherent only to one border of posteromedial commissure; feared insufficiency. Anterolateral opened

L. A.—posteromedial commissure heavily calcified; no fusion of leaflets anteriorly; posteromedial commissure opened

H. M.—heavily calcified and regurgitant posteromedial commissure; feared production of increased regurgitation if separated. Anterolateral opened

---

*Charles P. Bailey, Houck E. Bolton, Henry T. Nichols.

It must be realized that while minor pathologic changes may be found in the chordae of most patients with mitral stenosis, and while various degrees of agglutination or clumping of adjacent chordae supporting the same leaflet are seen frequently, cross-fusion of the chordae to the opposite leaflets in such a form as to cause a true subvalvular stenosis is relatively less common (Figs. 5, *A, B, C.*).

*Mitral insufficiency:* Should an element of mitral incompetence (perhaps previously entirely unsuspected) be found to exist, it should be evaluated as to amount and to site of localization with respect to the leaflet structure and the extremities of the line of valve closure. This information along with an appraisal of the condition of the leaflets (calcification, retraction, etc.) and the supporting chordopapillary system will furnish the basis for a decision as to whether a later operation[30] through a left-sided incision may be indicated (Fig. 6, *A, B, C*).

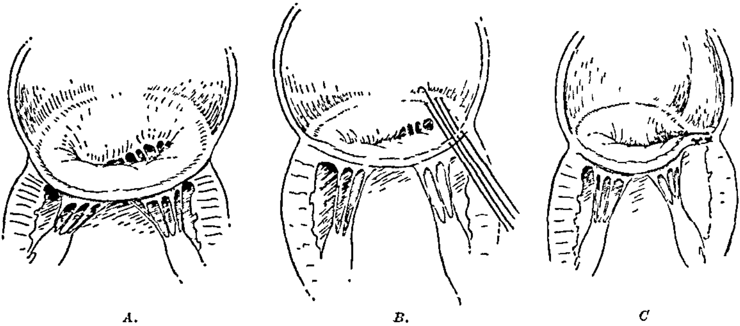


Fig. 6.—Nichols' method of polar cross-fusion of the annulus fibrosus for the correction of mitral insufficiency. (Courtesy New York J. Med., March 15, 1956.)

*A*, Typical posteriorly incompetent mitral valve with retraction of free margin of the posterior portion of mural leaflet. A similarly localized area of incompetence may be noted in cases of extreme annular dilatation.

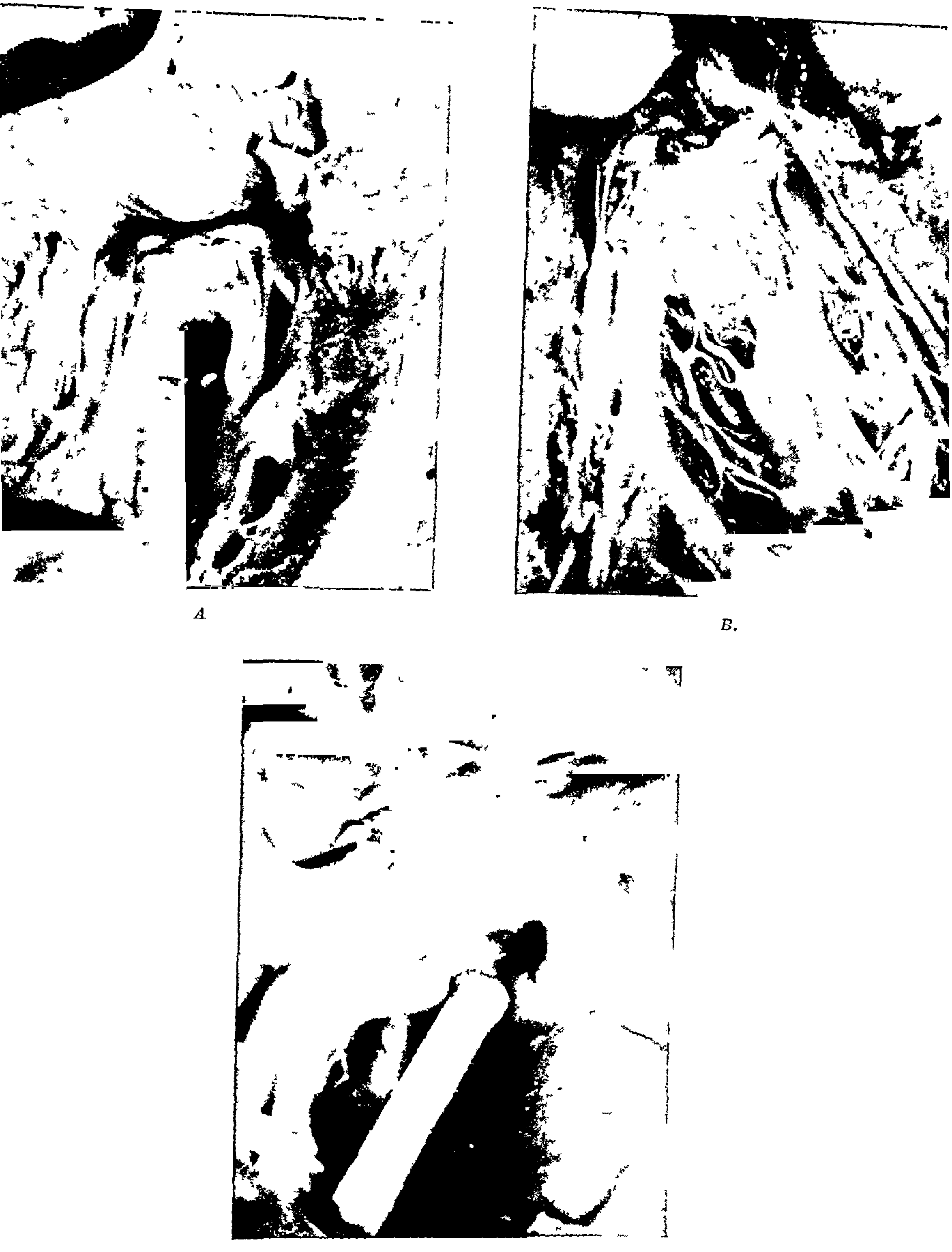
*B*, Plicating sutures placed about or through apposed portions of the annulus fibrosus across the incompetent pole of the valve, but preferably not actually over the valve aperture.

*C*, Tying down these sutures brings the selected portions of the annulus fibrosus into intimate early and, by healing, permanent contact. This reduces the over-all size of the area bounded by the annular ring, and by bringing the attached basal portions of the retracted leaflets closer together permits their free margins to come into full approximation during ventricular systole.

## MULTIVALVULAR INVOLVEMENT

The predominant tendency toward localization of the chronic rheumatic valvular lesions, as indicated in Table II, evidently is upon the mitral valve, but aortic involvement is a reasonable second, and tricuspid affection is far from infrequent. Apparently, the tricuspid valve never is found to be stenotic in the absence of a similar lesion of the mitral valve. However, isolated disease of either the aortic or mitral valve is seen not uncommonly.

Although the pulmonary valve, early in the course of rheumatic disease, exhibits classical rheumatic microscopic lesions involving the annulus fibrosus, it almost never develops the late features of leaflet thickening and commissural obliteration which are so characteristic of the rheumatic process.



A

B.

C.

Fig. 5—*A*, Marked thickening and partial fusion of adjacent chordae which does not cause significant subvalvular obstruction and which may be seen in many stenotic rheumatic mitral valves

*B*, Extensive subvalvular chordal change and cross-fusion which does substantially reinforce the obstruction produced by the primary cross-adherence of the leaflets (Courtesy Bailey Surgery of the Heart, Lea & Febiger, p 509.)

*C*, Extreme subvalvular change with direct union of agglomerate chordopapillary pillar-like masses with the apex of the valve funnel which has become elongated by the organization of fibrinous exudate deposited upon the free margins of the thickened leaflets Here the subvalvular pathology is extreme and will require definitive separation, perhaps by instrumental means, in order to relieve the circulatory obstruction (Courtesy Surg. Clin North

Except in the case of those relatively few patients with combined aortic and mitral stenosis who present an equivocal or no pressure gradient across the aortic valve, we believe that the surgeon who undertakes to operate for dynamically significant aortic stenosis logically should attack this valve before the mitral, using an appropriate thoracic incision (now nearly always the right third anterior interspace). Having completed the aortic surgery up to and including the suture repair of the aortic wall, he then will explore the mitral valve digitally if there is any physiologic (catheterization), auscultatory, or other evidence (such as atrial fibrillation) which is suggestive of mitral valve disease.

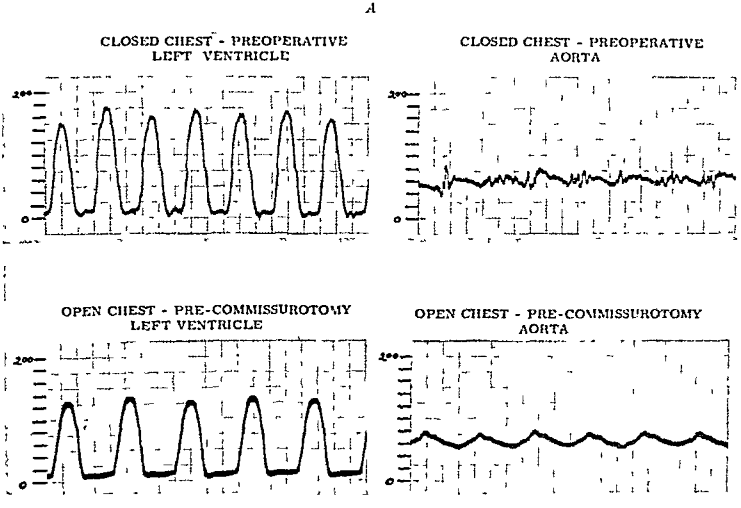


Fig. 7.—"Physiological damping" of the pressure curves obtained by direct needle puncture respectively within the aorta and the left ventricle during the course of surgery (Courtesy H. Goldberg, Brith Sholom Cardiopulmonary Laboratory, Philadelphia, Pa.)

*A*, Pressure curves obtained preoperatively by left heart catheterization in Patient W. R. with severe aortic stenosis. Note the large systolic gradient across the aortic valve.

*B*, Similar tracings obtained in the same patient by direct puncture of the aorta and left ventricle at the time of operation prior to the definitive aortic commissurotomy. Note the damping of the curves and the reduction in the gradient.

TRICUSPID STENOSIS.—In contrast to the situation which pertains with combined aortic and mitral stenosis in which at least the proper diagnosis nearly always may be made from the characteristic physical and physiologic findings, it should be emphasized that serious degrees of tricuspid stenosis or regurgitation frequently may remain unsuspected even after extensive clinical physiologic investigation; the effects of these lesions being largely obs

It must be recognized, of course, that considerable pathologic alteration of a valve must take place before a clinical effect is recognizable. Minor degrees of leaflet thickening and beginning commissural fusion may produce auscultatory changes long before significant effects are imparted to the circulation. For severe disturbances of physiology to develop more profound pathologic changes necessarily must take place.

AORTIC STENOSIS.—The logical approach to correction of a stenotic lesion of the aortic valve has been shown to be[31-34] by way of a pouch sutured to the ascending aorta, using a thoracic incision made through the right third or fourth interspace, anteriorly. It must be appreciated that the newer methods of evaluation of aortic stenosis (left heart catheterization; preferably combined simultaneous catheterization of the right side of the heart to determine the cardiac output by the Fick principle and of the left side of the heart to determine the pressure gradient across the valve) permit a rather exact preoperative determination of the amount and degree of physiologic obstruction of this valve which may be present. Hence, it should rarely be considered necessary to explore the aortic valve digitally for the purpose of making an exact diagnosis. The preoperatively determined systolic gradient which exists across this valve gives one the essential criterion upon which to base a decision for or against definitive intervention. It is felt that a systolic gradient of 40 mm. Hg across the aortic valve (or more), in isolated aortic stenosis, is necessary to justify aortic exploration.

*Aggravation of the Effects of Aortic Stenosis Following Mitral Commissurotomy.*—It is realized that in certain cases of combined mitral and aortic stenosis, the obstruction at the level of the mitral valve may be so severe that the relatively small amount of blood which passes through it readily can traverse even a fairly small aortic valve orifice without creating a significant pressure gradient across this valve. In such an individual, surgical relief of the mitral obstruction may produce such an increase in the blood flow through the aortic valve that a much larger pressure gradient (systolic) may develop across it, postoperatively. Direct measurement of this aortic gradient at operation before and after the mitral valve has been opened has not proved to be completely satisfactory, although it is employed in all doubtful cases. Apparently, this is due to a reduction in the cardiac output due to anesthesia, thoracotomy, opening of the pericardium, and operative blood loss (Fig. 7, *A*, *B*). This may be considered a "physiologic damping" of the pressure curves. However, failure to relieve a severe coexisting aortic stenosis at the same time that a mitral obstruction is corrected constitutes a major disservice to the patient since isolated aortic stenosis runs a more rapid devolutionary course than does combined mitral and aortic stenosis of equal severity. In our early experience, we have had several patients in whom a mild thrill over the aorta, at the time of surgery, was adjudged insufficiently pronounced to indicate aortic surgery. A few of these patients not only failed to improve following mitral commissurotomy, but went on later to die of their aortic disease.

many of the individual patients would have contributed to an unsatisfactory or to an incompletely satisfactory clinical result. These considerations have impressed us with the necessity for routine digital exploration of the tricuspid valve in every patient who is operated for mitral stenosis, provided there is no contraindication.

TABLE VII, B. DEGREE OF TRICUSPID INSUFFICIENCY OCCURRING IN 175 CASES OF TRICUSPID EXPLORATION AMONG 200 CONSECUTIVE MITRAL COMMISSUROTOMIES FROM THE RIGHT SIDE

| | NUMBER OF CASES |
|---|---|
| *Not Associated With Stenosis* | |
| Severe insufficiency* (3 plus) | 3 |
| Moderate insufficiency (2 plus) | 3 |
| Minimal insufficiency (1 plus) | 14 |
| With Stenosis (Before Tricuspid Commissurotomy) | |
| Severe insufficiency (3 plus) | 0 |
| Moderate insufficiency† (2 plus) | 3 |
| Minimal insufficiency† (1 plus) | 11 |
| Total | 4 |

*Tricuspid annulus fibrosus plicated in [illegible] patients.
†Included in Table VII A.

TRICUSPID REGURGITATION.—Tricuspid insufficiency, fortunately, is an abnormality which physiologically is less difficult to bear than are equivalent regurgitant lesions of the other two valves which are subject to rheumatic deformation (aortic and mitral). Nevertheless, in its more severe forms it is a distinct burden to the patient, especially when it is found in one with increased pulmonary arteriolar resistance, a condition often associated with (and caused by) mitral stenosis. With such increased vascular resistance effective function of the right ventricle becomes especially important in order to propel the blood through the pulmonary vascular bed. This is accomplished with great difficulty in the presence of a seriously incompetent tricuspid valve since during systole the right ventricular content more easily may be delivered in retrograde fashion through this valve into the systemic venous bed rather than into the pulmonary artery.

The correction of tricuspid insufficiency by direct, digitally controlled plication of the external portion of the tricuspid annulus fibrosus, as previously described,[36] may be carried out expeditiously and almost without significant additional risk through the right anterior thoracic approach (Fig. 8, *A-E*). However, it can be performed only with difficulty if the incision has been made at a level higher than the right fourth interspace (the fifth is preferable). In 2 of these patients, such plication effectively corrected the regurgitation. We believe this procedure to be indicated whenever the regurgitation is estimated (digitally) to be of a magnitude greater than 2 plus, providing, of course, that the patient's general condition is deemed satisfactory for such additional surgery. Recently, it has been possible to overcome severe isolated tricuspid insufficiency in one patient (D. C.) by the Nichols'[30] method of point cross plication of the anterolateral portion of the tricuspid annulus fibrosus (Fig. 9, *A-C*).

by the more obvious manifestations of the ever-present associated mitral disease. Although Reale, and his co-workers[35] have described well the physiologic findings which are characteristic of its presence, in practice, significant tricuspid disease can be recognized with certainty only by direct digital exploration of the tricuspid valve at time of mitral surgery (Table VII, A).

Table VII, A. Degree of Tricuspid Stenosis Found in 175* Cases of Tricuspid Exploration Among 200 Consecutive Mitral Commissurotomies From the Right Side

| | | TOTAL | PER CENT |
|---|---|---|---|
| Severe Stenosis | | 11 | 6.2 |
| Finger-tip opening | 3 | | |
| 1 finger opening (1.5 cm.$^2$) | 8 | | |
| Moderate Stenosis | | 8 | 4.5 |
| 1¼ finger opening | 3 | | |
| 1½ finger opening (2.5 cm.$^2$) | 5 | | |
| Minimal Stenosis | | 21 | 12.0 |
| 1¾ finger opening | 3 | | |
| 2 finger opening | 10 | | |
| 2½ finger opening | 4 | | |
| Unspecified minimal involvement | 4 | | |
| Total | | 40 | 22.7 |

*In 25 patients tricuspid exploration was not carried out.

*Exploration of Tricuspid Valve.*—Insertion of the operator's finger through an incision made in the right auricular appendage to explore the right atrial chamber when using a left-sided approach is difficult and dangerous. We employed it in 7 patients, the first time on Jan. 23, 1953, and described it only to condemn it.[16] Any attempt at instrumentation of this valve from the left is fraught with unusual danger due to the poor exposure and control of the appendage. In truth, this is an attempt to perform a right-sided operation through a left-sided approach!! Tricuspid commissurotomy performed through a right thoracic incision, on the other hand, is readily, efficiently, and safely accomplished. If we are to include a routine exploration of the tricuspid valve with every operation for mitral stenosis, the approach must be made from the right.

Among the 200 patients, 30 were operated on primarily for aortic lesions. There were 170 patients who were operated on for a diagnosed mitral stenosis, although in 2 an additional preoperative diagnosis of tricuspid regurgitation, and in 5 of tricuspid stenosis, had been made. These suspected additional valvular lesions were found, and also another fourteen instances of significant (moderate to severe) tricuspid stenosis (Table VII, A), as well as another 7 cases of previously unsuspected significant incompetency of the tricuspid valve (Table VII, B) in 175 of these patients[35] in whom the tricuspid valve was explored. In 25, hypotension or other untoward signs precluded elective examination of this valve. Had we failed to perform digital examination of the interior of the right atrium in the majority of this group of patients, the multivalvular involvement in most cases would have escaped notice and in

APPENDAGEAL THROMBOSIS.—Since dislodgment of loosely attached thrombotic material from the appendage (atrial thrombosis was present in 29 per cent of our first 1,200 patients with mitral stenosis in whom commissurotomy was performed from the left side) during the entrance of the operating finger or during the subsequent intracardiac manipulations is believed to be the chief and usual cause of systemic arterial embolization when it occurs during surgery (Table VIII), one logically might expect to eliminate such accidents from this source by choosing a different site of atrial entrance. Using the dissected interatrial groove as the approach the entrance is made through the left side of the interatrial septum, a site characteristically free of thrombotic attachment. As previously reported,[16, 37] with the routine application of transient constriction to the carotid vessels when the left atrial lumen is entered via the left auricular appendage, the incidence of cerebral embolization occurring at the time of surgery can be reduced from 5.1 per cent to 1.8 per cent. However, since this technique merely redirects the course of any dislodged particulate matter into the descending aorta rather than through the carotid arteries, the incidence of peripheral arterial embolization becomes increased correspondingly.[1]

TRUE ATRIAL THROMBOSIS.—Large extensions of thrombosis from the left auricular appendage into the true atrial lumen were recognized in 3.9 per cent of our first 1,200 patients (operated on from the left). In most instances, the clot forms in "placenta like" fashion, coming partially to line the left or posterior wall of the left atrium. Usually such "lining" clots are found in an organized state and are well attached.

However, in some patients, massive clotting occupying up to one half or more of the volume of the left atrial chamber may be encountered. If the operator's finger, after recognizing this situation is kept carefully away from such (usually soft) masses, apparently there is little danger of dislodgment of a portion.

Extensive intra-atrial thrombosis was recognized at surgery in 16 of our 200 patients (8 per cent) operated upon from the right side.

Occasionally, the operator may recognize the presence of a soft freely floating thrombotic mass within the left atrial chamber. In still others, a similar mass may be found to be attached loosely, perhaps by a thin pedicle, and capable of moving freely within a wide arc. Either of these types of "loose" thrombus may lead to the "ball valve" syndrome with intermittent total blockage of the diminutive mitral valve orifice.[16, 38] On March 21, 1955, in Patient L. G. (Table VIII), a soft, loosely attached clot, measuring approximately 3 cm. in diameter, became detached during the surgical manipulations incident to enlargement of the valve aperture, and thereafter disappeared from the left atrial chamber. Immediate palpation of both femoral arteries revealed disappearance of pulsations. Upon completion of the thoracic surgery, an abdominal incision was made and the embolic mass was removed from the aortic lumen close to its bifurcation. Recovery was uneventful.

Such poorly attached or floating clots sometimes may be evacuated from the momentarily opened heart by a technique of permitting the pressure of

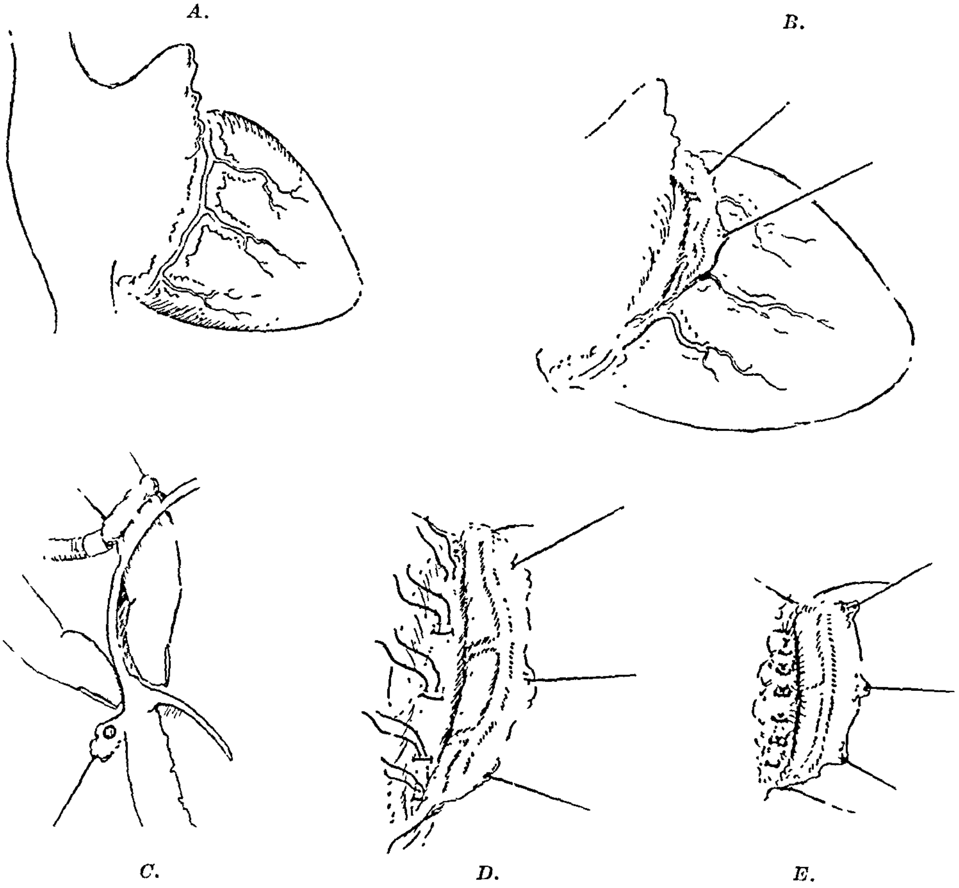


Fig. 8.—Partial circumferential plication of the mural portion of the tricuspid annulus fibrosus for insufficiency of this valve. (Courtesy Bailey, et al., Cardiovascular Surgery, Proceedings, Henry Ford Hospital Symposium, 1955, W. B. Saunders Company.)

*A*, Right atrioventricular sulcus.

*B*, Mobilization of fat pad and contained right circumflex coronary artery *from the sulcus*.

*C*, Left index finger inserted into right atrium through incision made in right atrial appendage and thumb placed beneath dissected fat pad (and right circumflex coronary artery) to palpate annular ring.

*D*, Placement of interlocking heavy mattress sutures into and about a portion of the perimeter of the valve ring.

*E*, Tying down of these sutures effectively reduces the length of this portion of the ring.

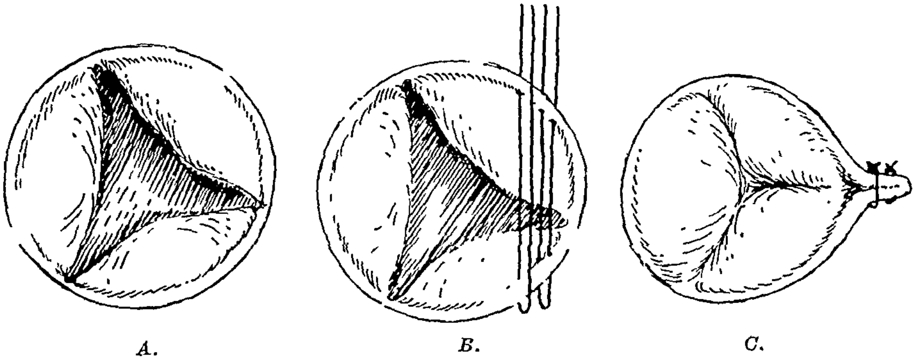


Fig. 9.—Nichols' method of polar cross-plication of the annulus fibrosus applied in tricuspid regurgitation.

A, Preoperative condition of the valve with either a dilated ring or shrunken leaflets resulting in a significant disproportion between the available existing leaflets substance and the size of the aperture to be defended.

*B*, Placement of plicating sutures in the annulus over the anterolateral extremity of the triangular valve orifice.

*C*, Incompetence of the valve is markedly reduced or overcome as the sutures are tied down and the bases of two of the leaflets are brought closer together.

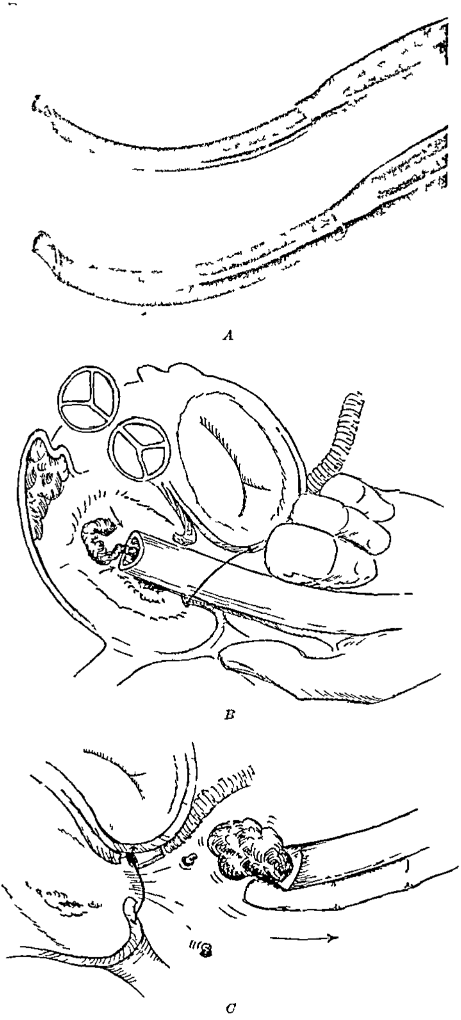


Fig 10—Use of one of the Litwak* aspirating tips to remove a loosely attached or freely floating thrombus from the left atrial chamber (right sided approach)

*A* Photograph showing two sizes of the instrument (Courtesy New York J Med March 15 1956)

*B* Introduction of streamlined instrument along the palmar surface of the intracardiac finger from the right (it is used similarly from the left) The suction is applied after the tip of the aspirator has been brought into contact with the clot (Courtesy New York J Med March 15 1956)

*C* Sudden removal of the finger the instrument and the attached clot from the heart *en masse* The concomitant momentary free bleeding from the heart wound serves to out any smaller fragments which may have become detached during the extraction

---

*Obtainable from George P Pilling & Son Co Philadelphia

TABLE VIII. OPERATIVE EMBOLIZATION AMONG THE FIRST 200 MITRAL COMMISSUROTOMIES FROM THE RIGHT SIDE

| PATIENT | PROCEDURE | CLOT IN ATRIUM | CALCIFICATION IN LEAFLETS | TO | OUTCOME |
|---|---|---|---|---|---|
| P. R. | Mitral comm., aortic explored | Large, soft granular | Posterior commissure and septal leaflet | Brain | Survived with alexia and agraphia only |
| L. G. | Mitral comm., tricuspid comm. | 3 cm. in diameter, above ant. comm. | Moderate | Aortic bifurcation | Survived; removed at same operation; no complications |
| M. L. | Mitral re-operation, tricuspid explored | Large, soft | Slight | Midbrain | Survived; diplopia only; improving |
| W. B. | Mitral comm., tricuspid explored | Numerous small vegetation-like clots | Moderate | Right femoral artery | Survived; complete relief with con. caudal block only; 3 days |
| M. M. | Mitral comm. | None | Embolus believed of calcific origin; periorificial nodular calcification located diffusely around all edges of both leaflets and commissures; felt to be loose during surgery | Brain | Died 24 hours; spastic biplegia; no recovery |

aorta immediately but may come to rest temporarily upon the inner surface of the most dependent wall of the left ventricle or atrium. From the latter site, they may be extracted by sliding them over to the site of atrial entrance with the ungloved index finger[11] (Fig 11, *A*, *B*, *C*).

As shown in Table VIII, all but one of our five instances of operatively produced embolization were of thrombotic origin.

### SURGERY FOR "RECURRENT" MITRAL STENOSIS

Obviously, a ready surgical approach from the right side would avoid many of the technical difficulties inherently associated with operation for mitral stenosis for the second time from the left. In contrast to a nearly virginal operative field on the right (except for pericardial adhesions), the left side then is encumbered by the presence of pleuropulmonary adhesions which tend to be very firm over the site of the previous pleural and pericardial incisions. Not only does the pericardium tend to be densely adherent to the stump of the partially amputated appendage, but the latter usually will be found to have become shrunken and imbedded within dense fibrous connective tissue. The important anatomic markings which normally delineate the limits of safe entry into the left atrial chamber are obscured. Hence, in these individuals the right-sided approach to the mitral valve may be made safely and methodically, while the left is fraught with unusual technical problems and the danger of causing air leaks and prolonged oozing from the scarred pleural surfaces, as well as increased real and fancied difficulties in separating the valve commissures. Not infrequently an unsatisfactory valve mobilization may result from hasty manipulations necessitated by insecure hemostatic control during secondary procedures carried out by the left-sided route.

### CASE REPORT

F. B. (Case No. 36918), a 23 year old white woman was operated upon by one of us (C. P. B.) for mitral stenosis at the Hahnemann Hospital, in Philadelphia, on Feb. 27, 1950. She was the fifteenth patient operated on at this hospital for this condition. The approach was made through an opening in the tip of the left auricular appendage, using the usual long posterolateral thoracic incision and opening the fourth intercostal space widely. The double glove technique then in vogue was employed.

The valve was found to be fibrosed but not calcified, and the orifice was contracted to a tiny slitlike aperture less than a centimeter in length and perhaps one fourth of that distance in width. There was no detectable regurgitation (Fig 12, *A*).

The screw type guillotine knife was applied against the anterolateral valve commissure and it was cut once. This separation was extended readily and fully to the annulus fibrosus by digital pressure without producing any regurgitation. Palpation with the finger tip revealed adequate support with numerous chordae attaching to both leaflets. It was estimated that the valve opening had been extended more than 1 cm. beyond the line of direct tendinous support.[17]

The operator's attention was turned toward the posteromedial commissure and digital pressure was made against it. At first it resisted strongly. The guillotine knife was closed once. Digital pressure was applied and suddenly the gloved finger seemed to break through the valve substance, separating the tissues widely. The resulting opening surely would have accommodated 3 fingers simultaneously. Immediately, a 4 plus grade of regurgitation became apparent, although palpation of the posterior portion of the valve opening vealed that there were several chordae attached to each the left atrial blood content to force it out, as described by Jamison,[39] and others. However, this maneuver may fail, especially if the mass is large or partially adherent to the endocardial surface.

In such a case, the end of an appropriately shaped, specially designed aspirating tip such as that devised by Litwak (Figs. 10, *A*, *B*, *C*) may be inserted into the atrium along with the finger until the end is in contact with the clot. The suction then is turned on and the instrument with attached clot (and finger) is withdrawn through the atrial site of entrance as the purse-string suture is relaxed momentarily. We have performed this maneuver successfully from the right side on three occasions.

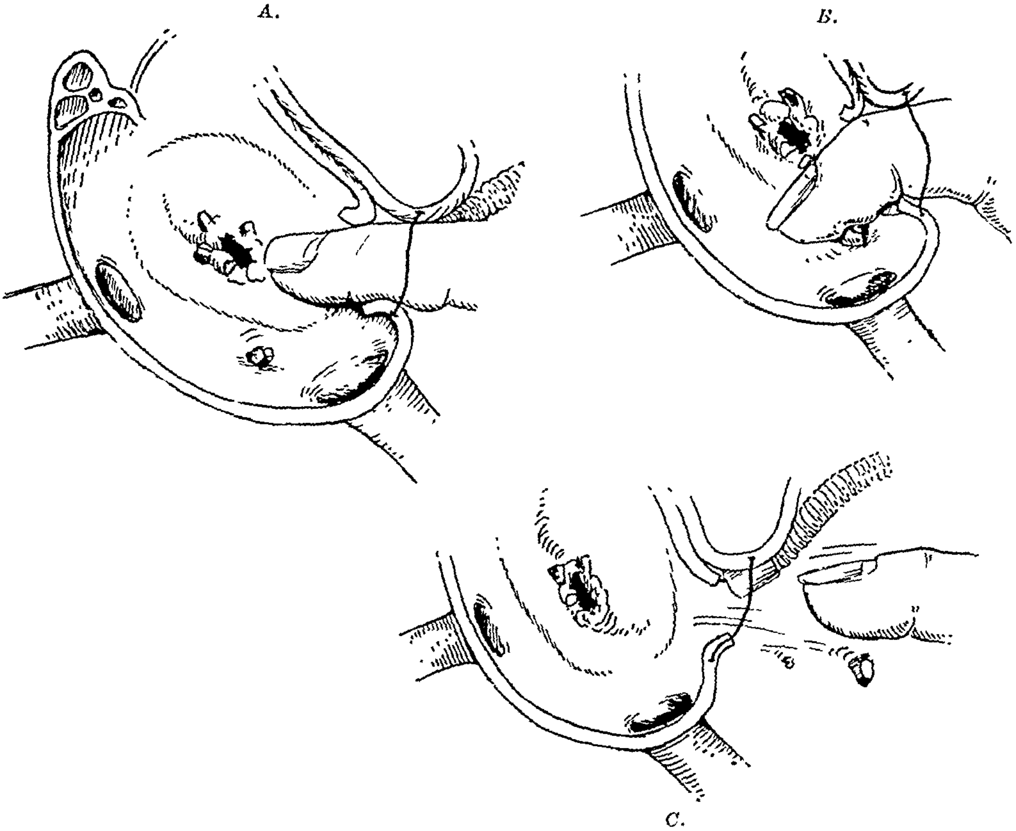


Fig. 11.—Extraction of dislodged calcific particles from the left atrium.

*A*, Calcific fragments being heavier than blood, when detached from the atrial aspect of the mitral valve by the surgeon's finger or instrument, tend to fall into and to rest for a while upon the most dependent wall of the left atrium.

*B*, When found by the bare index finger tip of the surgeon, such a particle may be held firmly against the internal surface of the atrium and then "slid" along it to the site of digital entrance.

*C*, The finger and particle together are removed suddenly from the heart, permitting momentary external bleeding. This tends to "flush out" any overlooked additional particulate matter. Hemostasis is re-established promptly by tightening the purse-string suture.

CALCIFICATION OF THE VALVE AS A SOURCE OF EMBOLI.—Naturally, fragmentation of calcific material which may be attached to the valve edges is not influenced by the choice of the site of entrance into the left atrium but only by the condition of the valve and by the manipulations entailed in the particular commissurotomy technique employed. Since such dislodged calcific particles are heavier than the blood, they may not be expelled into the

Operation was performed on April 23, 1953. Again the approach was made through the left thorax, the old incisional scar being opened. Great difficulty was experienced in mobilizing the left lung from the thoracic wall and the myocardium to which it was extensively adherent. Numerous small lacerations of the lung were produced with resulting air leaks.

Even before entering the extremely dilated left atrium, ventricular fibrillation developed once. Spontaneous rhythm was reestablished by massage, electric countershock, and the administration of vasopressive drugs. Nevertheless the blood pressure remained at a low level. Consultation with the family resulted in a decision to proceed with the surgery in view of her generally downhill course and the improbability of survival from a nonhelpful exploratory operation. Four subsequent episodes of ventricular fibrillation punctuated the ensuing operative procedure, each requiring electric countershock.

A purse-string suture was placed in the atrial wall at the base of the largely obliterated appendage and a stab incision was made within its confines. The operator's bare index finger was inserted into the atrium and the valve was examined. A 2½ to 3 plus regurgitation was present, but a remarkable change had taken place within the valve structures. The anterior commissure had become readherent all the way back to the anterior line of direct tendinous support. Posteriorly, the valve separation no longer extended to the annulus fibrosus, but scarcely a centimeter beyond the posterior line of direct tendinous support which could be delineated clearly with the bare finger. The entire valve aperture was considered to be a 1½ finger size. The obliterated anterior commissural tissue now was definitely calcified (Fig. 12, *C*).

At this point, ventricular fibrillation occurred again, requiring massage and stimulation.

The anterior commissural tissues were split readily to the valve ring without altering the existing regurgitation.

Ventricular fibrillation recurred, requiring electric countershock and massage for its correction.

Using the Jamison stitcher, a strip of pericardium was passed in suture fashion through puncture wounds made in both leaflets at points just beyond (posterior to) the posterior line of direct tendinous support. This technique has been described previously.[16,40] One end of the pericardial loop was passed through an "eye" made in the other, and the end of the loop thus created was drawn out of the atrial incision. Moderate traction upon this loop significantly diminished the regurgitant jet, leaving an effective valve opening more than sufficient to admit 2 fingers (Fig. 12, *D*).

The procedure was terminated by suspending the end of the pericardial loop from the atrial incision which was closed with cross-suturing of a mattress type. The chest wall was closed in the usual manner with water-seal drainage.

Ventricular fibrillation ensued twice during the closure of the chest wall.

The patient's blood pressure remained at a very low level (60 to 70 mm. Hg, systolic) in spite of transfusions and vasopressive agents, and she failed to react from the anesthetic. Twelve hours after the completion of the operation, the heartbeat disappeared.

Post-mortem examination confirmed the operative findings plus evidence of pulmonary edema and old infarcts of the spleen and left kidney.

*Comment.*—The clear-cut digital evidence of extensive re-establishment of commissural fusion both anterolaterally and posteromedially in this case, following a more than adequate bicommissural mobilization of the leaflets, leaves no room for doubt that mitral restenosis sometimes will take place despite the most extensive technical surgical mobilization of the valve.

### COMPLICATING PLEUROPULMONARY LESIONS

To less extent, the mentioned difficulties may apply in patients who previously have had left-sided thoracic surgery for some other reason, and in those posterior separation had become extended at least 2 cm. beyond the posterior line of direct tendinous support and fully to the annulus fibrosus. Presumably, it extended well beyond all normal chordal support and, therefore, even though apparently made in the proper anatomic line, permitted regurgitation due to reflux of the nonsupported portion of the flexible valve edges (Fig. 12, *B*).

The patient's heart dilated enormously, became visibly weaker in its contractions, and went into arrest. The finger was removed from the atrium, the appendage was ligated, and cardiac massage was commenced. With the administration of adrenalin and continued rhythmic compression of the ventricles, the heartbeat returned and a peripheral pulse and blood pressure became obtainable.

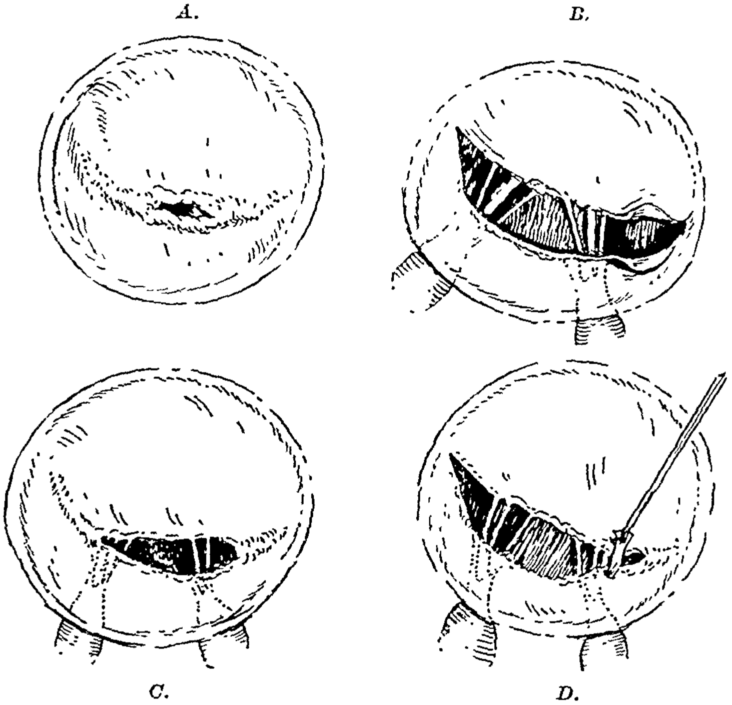


Fig. 12.—Diagrammatic representation of mitral valve in patient (F. B.) demonstrating the significant degree of restenosis which occurred within a five-year period following extensive bicommissural mobilization of the structures.

*A*, Original stenotic valve orifice in a fibrosed but largely flexible valve.

*B*, Following an extensive commissurotomy at each valve extremity, a very severe posteriorly located regurgitant jet was noted (estimated to be of 4 plus magnitude).

*C*, Five years later, at reoperation, the anterolateral commissure had become obliterated to the anterior line of direct tendinous support, and the posteromedial had become refused partially. The regurgitation had become reduced significantly.

*D*, Direct suturing together of the leaflets over the incompetent pole of the valve reduced the regurgitant jet. The anterolateral commissure had been reopened.

The chest wall was closed with some difficulty because of the great dilatation of the left ventricle which scarcely could be fitted within the thoracic cage. It was impossible to approximate the edges of the widely opened pericardium so the sac was left entirely open.

While the patient remained hypotensive for days, eventually she made a reasonably satisfactory recovery, although the heart showed marked enlargement and there was an entirely new 4 plus systolic murmur over the apex. The previously present diastolic murmur was entirely gone. Atrial fibrillation persisted.

During the next four years, the patient continued to present evidence of mitral insufficiency with a lowered myocardial reserve, tendency to edema of the lower extremities, and engorgement of the liver.

On April 3, 1955, this patient was readmitted to the Hahnemann Hospital (Case No. 55-04017) for repair of the mitral insufficiency. Cardiac enlargement was 3 plus and the left atrium was so enlarged as to be considered aneurysmal.

even a degree of scoliosis may follow. While early and persistent attention to movement tends greatly to diminish the incidence of these complications, every thoracic surgeon has had experience with at least a few individuals in whom these efforts have failed, a permanently stiff and painful shoulder resulting.

In striking contrast, such shoulder complications are almost never seen when an anterolateral thoracic incision is employed.

### DIVISION OF THORACIC MUSCLE TRUNKS

The anterolateral incision divides relatively few fibers of the pectoralis major muscle. By a splitting technique, the fibers of the lower third of the muscle mass are separated anatomically over the interspace selected for thoracic entrance, but their innervation, which comes from the axillary region by way of the middle and lateral anterior thoracic nerves, is not interrupted. Hence, the function of this muscle ultimately becomes restored nearly completely to normal.

In dismal contrast, the usual long posterolateral thoracic incision necessarily divides the continuity of the latissimus dorsi completely. At the same time, it denervates the caudal portion of this muscle permanently since it entails division of the spinal accessory nerve filaments which run over and through the muscle structure roughly parallel with the course of its fibers. The rhomboidei and the serratus magnus muscles suffer serious but less extensive damage.

It would seem inevitable that a significant portion of the muscular strength of the shoulder and arm must be destroyed by this type of incision. While such a reduction in the structural strength of the arm, shoulder, and trunk may be of little consequence in a person of sedentary habits, in one accustomed to greater physical activity and vigor, it may reduce in value the benefit obtained from a successful attack upon the valve by precluding a return to his previous vocation or avocations.

What part this weakening of the muscular support may play in the postoperative development of a stiff shoulder has not been determined.

### COSMETIC ASPECTS OF THE INCISION

An apparently minor advantage of the anterolateral incision is related to its placement (in the female) largely within or slightly below the inframammary fold. This localization renders the incisional scar scarcely visible in the nude, and completely concealed when the patient wears the usual type of breast support or a modern two-piece bathing suit (Fig. 13, *A* and *B*).

In 25 consecutive female patients operated on through a right-sided incision, the length of the incision was found to average 27 cm. A comparison was made with a similar group of patients in whom the approach to the mitral valve had been made through a left posterolateral wound. The average length of the scars measured 47 cm.

Inasmuch as 71.5 per cent of the patients in whom we have performed commissurotomy for isolated mitral stenosis are females, and since 75.7 per cent of these were below the age of 45, it may not be amiss to consider who have suffered, on a former occasion, from left pleural effusion or empyema, or perhaps even from extensive (left) pulmonary disease such as bronchiectasis, abscess, or tuberculosis. The right-sided approach to the mitral valve makes it possible to by-pass such difficulties in some cases, and with other individuals to undertake simultaneous corrective surgery when indicated for right-sided noncardiac associated lesions.[28] This applies especially to decortication of organized exudate which may form upon the right lung when it has been compressed over a long period of time by pleural effusion which may have been of cardiac origin (failure).

### INTERATRIAL SEPTAL DEFECT

We have encountered one small, previously undiagnosed, interatrial septal defect which we were able to close by the technique of atrio-septo-pexy at the time of the right-sided operation for mitral stenosis, as well as two others which were diagnosed correctly, preoperatively. In these latter instances, the mitral valve was approached by way of the right auricular appendage and the septal defect.[41, 42]

### ANOMALOUS PULMONARY VENOUS DRAINAGE

Right-sided anomalies of the pulmonary venous drainage may be diagnosed and treated appropriately during the course of the routine exploration of the right atrium. In one of the patients, with interatrial septal defect referred to above, an associated anomalous right pulmonary vein was ransposed with the help of the left index finger inserted within the right trial chamber by a technique described previously.[16, 18]

### INCISIONAL PAIN

One of the lesser but nevertheless worth-while advantages of the right-ided thoracic incision is the relative mildness of the thoracic pain experienced during the postoperative period. This was dramatically illustrated in the reports of each of the 6 patients who had secondary surgery performed from the right after previously undergoing left-sided mitral surgery. It must be noted that this minimization of discomfort is equally prominent when the left anterolateral thoracic incision is employed for mitral commissurotomy and merely represents the inherent superiority of the anterolateral over the posterolateral thoracic incisions in this respect.

### ARM AND SHOULDER FUNCTION

In part, the prolonged limitation and stiffness of movements of the shoulder girdle which sometimes follow surgical procedures which utilize posterolateral thoracic incisions are related to the initial operative pain and consequent muscle spasm. The voluntary immobilization of these structures which often is established during the immediate postoperative period, may lead to adhesion formation about the shoulder joint, to bursitis, or to a habit of maintained chronic muscular tension which tends permanently to inhibit free movement of the shoulder girdle. A high-held or frozen shoulder and

THE THORACIC ENTRANCE.—In the female, a smoothly curved incision is made 1 cm. below the right inframammary fold from the sternal border at the level of the fourth costal cartilage to within 4 cm. of the apex of the axilla. In the male, the incision is made somewhat less curved and passes just below the nipple. The fibers of the pectoralis major and minor are split and divided as necessary to expose the fourth intercostal space. The long thoracic nerve running longitudinally close to the midaxillary line is preserved. The intercostal muscles are incised in the middle of the interspace, and the right pleural cavity is entered. The fourth costal cartilage may be divided at its sternal junction to afford better exposure of the intrathoracic contents. The internal mammary artery and veins are mobilized separately. At this level, it is common to find one vein medial and one vein lateral to the artery. Each vessel is doubly clamped, divided between the clamps and the cut ends are ligated individually. So treated, few of these vessels will bleed at a later time. The fourth and fifth ribs are covered with large, moist gauze pads and are spread apart to a limited extent by a Finochietto or other type of self-retaining retractor. To allow additional separation of the ribs, the intercostal muscles are divided farther posteriorly along with the pleura. Over a period of twenty minutes, the retractor spread is increased by small increments, thus avoiding rib fracture.

THE CARDIAC APPROACH.—The lung is compressed posteriorly with a large gauze pad *exposing the pericardium. This is incised in the pattern of the* capital letter "L" lying on its side. One limb of the pericardial incision runs parallel to the right phrenic nerve and 1 cm. anterior to it extending from the arch of the aorta superiorly to the lower margin of the right inferior pulmonary vein. The other limb is directed forward at 90 degrees from this latter point across the right atrium until it passes just under the sternal border at the anterior extremity of the wound. Care is taken to ligate all bleeding points along the pericardial edges. Traction sutures are placed in the anterior and posterior lips of the pericardium. The phrenic nerve is avoided assiduously since inadvertently produced diaphragmatic paralysis will result in postoperative persistence of the usually presenting symptom of shortness of breath.

A distinct groove or sulcus can be made out upon close inspection of the surface of the right side of the heart (Fig. 14, *A*). It runs in a cephalocaudad direction, starting just behind the superior vena cava and separating the right atrium, which lies anteriorly, from the left atrium along a line proximal to the junction of the right pulmonary veins. This interatrial sulcus is covered with epicardium and is filled in with adipose tissue so that it is best visualized during atrial systole. This is the same sulcus which is dissected in the Sondergaard technique for closure of interatrial septal defects.[43]

The interatrial boundary may be demonstrated more definitely by making a limited (1.5 to 2 cm.) longitudinal incision in the epicardium over the middle of this lightly colored fat pad, and by continuing this dissection bluntly to a depth of 0.5 cm. (Fig. 14, *B*).

seriously the cosmetic aspects of these operations. An incalculable amount of psychologic distress may be imposed upon a sensitive patient by the creation of an ugly or deforming scar.

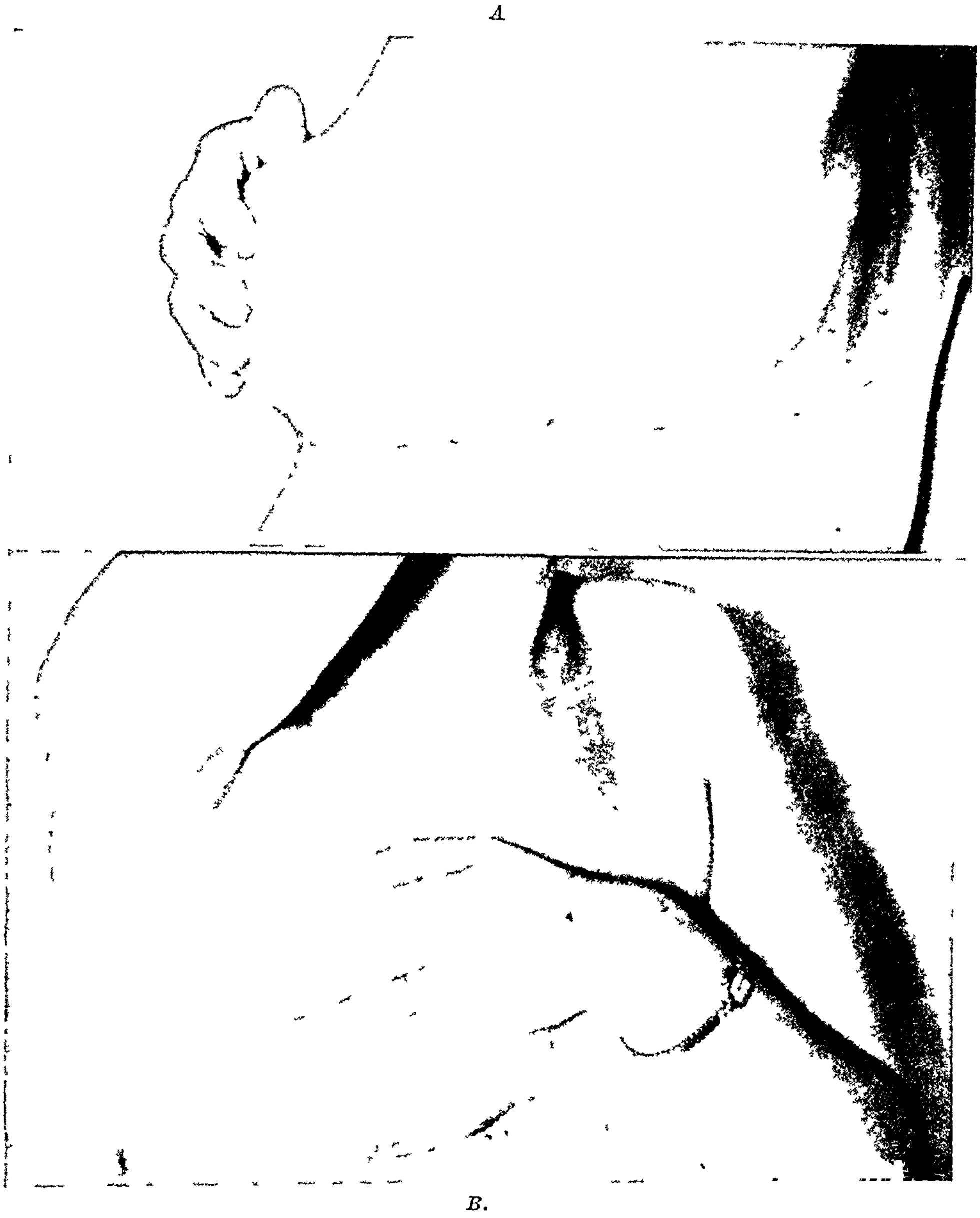


Fig. 13.—*A*, Long (average 47 cm.) posterolateral incisional scar resulting from customary left-sided operation for mitral stenosis.

*B*, Cosmetically superior and much shorter (27 cm.) anterolateral incisional scar produced during right-sided approach to the mitral valve. The transaortic approach for aortic stenosis and the natural approach to the tricuspid readily may be made through this same incision.

Courtesy New York J. Med., vol. 56, March, 1956.

## OPERATIVE TECHNIQUE FOR MITRAL COMMISSUROTOMY PERFORMED FROM THE RIGHT SIDE

The patient is positioned on his back in the supine position. A small pillow or folded sheet may be placed under the right shoulder to tilt the patient slightly toward the left, and the right arm is abducted 90 degrees.

groove may be extended and deepened somewhat to admit the tip of the operator's *left* index finger from which the glove finger has been cut away The bare finger is moistened with a dilute heparin (1/1000) solution and is inserted, palmar surface uppermost, into the apex of the dissection The end of a broad-bladed knife (Bard Parker No 22) is laid flat upon the palmar aspect of the distal phalanx of the left index finger Fig 15, *B* shows a special guard devised for the blade by Dr Richard A Gilman This instrument serves to minimize any danger of dividing the purse string and reduces the possibility of penetrating the right atrial chamber inadvertently This accident occurred three times in our early experience In each instance, the opening was closed securely with interrupted nylon sutures by a technique reminiscent of the atrio septo pexy procedure for interatrial septal defect[40]

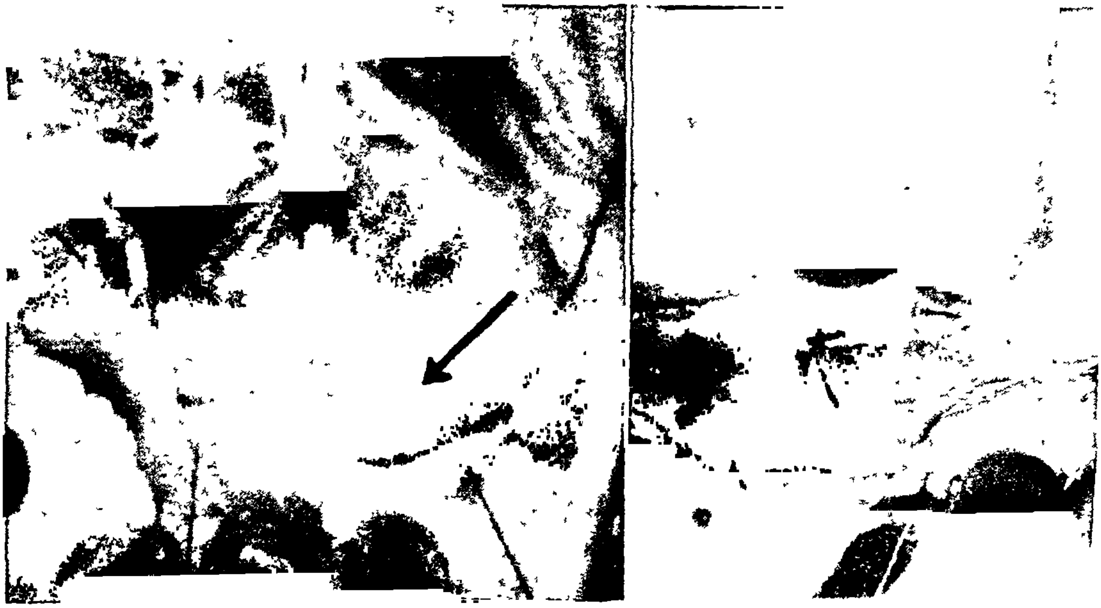
Controlled and guided by the finger, the blade is advanced to pierce the posterior half of the partially separated interatrial septum It is important to limit the extent to which the knife penetrates within the lumen of the left atrium Otherwise, there is some chance that it may puncture the posterior (vertebral) wall of this chamber (Fig 15, *C*, and *D*) and cause severe hemorrhage, the site of which cannot be exposed for suturing except by promptly transecting the sternum, opening the left pleural cavity, and dislocating the heart upward and out of the pericardial sac The Gilman guard, by limiting the length of blade tip which may emerge from its cover, would seem to obviate this risk

The entrance of the knife blade into the left atrial chamber is heralded *by a small gush of bright red blood The knife is withdrawn immediately and* the finger tip is inserted into the opening to tamponade it As the finger tip is advanced cautiously perhaps with a gentle twisting motion, the stab wound in the left hemiseptum gradually becomes dilated to accommodate it (Fig

TABLE IX HAZARDS OF RIGHT SIDED ENTRANCE INTO LEFT ATRIUM AND APPROPRIATE PREVENTIVE AND CORRECTIVE MEASURES

| HAZARDS | SAFEGUARDS AND MANAGEMENT |
|---|---|
| 1 Hypotension from pressure on right side of heart | 1 Close touch with anesthesiologist, avoidance of prolonged retraction against right atrium, use of vasopressive agents |
| 2 Tearing out of purse string (possible massive hemorrhage) | 2 Care to see that purse string loops penetrate full thickness of atrial wall, approximation of lips of cardiac wound with Nelson tissue forceps if this accident should occur |
| 3 Accidental cutting of purse string (possible massive hemorrhage) | 3 Use of Gilman guard (see Fig 15, *B*) on entering left atrium, approximation of lips of cardiac wound with Nelson tissue forceps |
| 4 Laceration *into right as well as left* atrium, production of septal defect | 4 Care *to cut into only posterior* half of dissected interatrial septum, repair of septal opening by the technique of atrio septo pexy |
| 5 Stab wound penetration of posterior wall of left atrium | 5 Care not to insert knife blade [illegible] nly (Gilman guard), pr[illegible] pl[illegible] [illegible] of to [illegible] |

THE ENTRANCE INTO THE LEFT ATRIUM.—A purse-string suture of heavy braided nylon (No. 2) swedged upon a curved needle is placed widely (at a distance of .5 to 1 cm.) about the dissected area. Care is taken that the internal loops are made less than 8 mm. in length, and that the needle point actually penetrates into the lumen of the underlying atrium with each pass. Failure to include the entire thickness of the atrial wall within the suture line will render the attachment of this hemostatic purse string insecure. It is essential also definitely to traverse the underlying interatrial septum with the purse-string suture at the upper and lower ends of the interatrial groove in order to limit any tendency toward "spontaneous" separation of the two halves of the laminated interatrial septum. Reinforcement of this restraining effect is accomplished by placing a No. 3-0 cotton stay suture deeply into and across the interatrial groove and its contents just beyond the purse-string suture at each end of the groove. The ends of these cotton sutures are left long after tying and the needles are permitted to remain attached so that they may be used later to oversew the dissected portion of the groove (Fig. 15, *A*).



*A.* *B.*

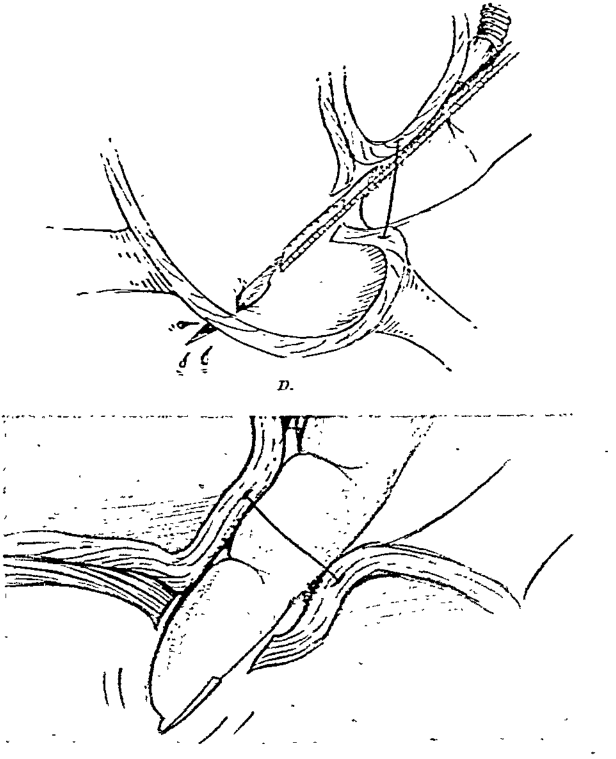
Fig. 14.—*A*, Photograph of the right side of the heart at operation, showing location of interatrial groove (arrow).

*B*, Photograph showing the limited dissection of the interatrial groove and placement of the hemostatic purse-string suture. (Courtesy of A.M.A. Book of Exhibits, 1955.)

The long ends of the nylon purse string now are incorporated within a Rumel-Belmont tourniquet in order to apply flexible, dependable, hemostatic tension to the suture line. A heavy braided nylon suture with a swedged-on curved needle is placed in mattress fashion across the area of dissection in such a way that the lips of the wound to be made in the left atrial wall must become everted when it is tied down. The incision will be made within the confines of this mattress suture, care being taken not to interrupt it. By this means no raw wound will be left finally within the left atrial chamber. The dissection of the

15, *E*). The technical hazards associated with the right-sided entrance into the left atrium are listed in Table IX. The treatment as well as the prevention of these errors is suggested.

EXPLORATION OF THE LEFT ATRIUM AND THE MITRAL VALVE.—The valve orifice when so approached lies somewhat farther from the site of cardiac entrance than when explored from the more customary left side. This may necessitate the insertion of the whole length of the finger into the atrial



*D.*

*E.*

Fig. 15.—*A*. Illustration showing placement of restraining stay sutures through the partially split atrial septum at either extremity of the dissection.

*B*. Heavy braided nylon mattress suture placed across dissected region in such a way that tying down will evert lips of left atrial incision (to be made).

*C*. Penetration of the lower (left) half of the dissected septum with knife blade. This incision is kept within the confines of the mattress suture.

*D*. Possible perforation of the posterior atrial wall by a simple knife blade if it is inserted too far.

*E*. Entrance of ungloved tip of left index finger through slitlike puncture opening made in left half of dissected septum. As the finger is advanced in a gentle twisting fashion, the septal incision becomes lengthened somewhat to accommodate it. Usually, tension upon the purse string is not required for hemostasis at this time. (Courtesy New York J. Med., March 15, 1956.)

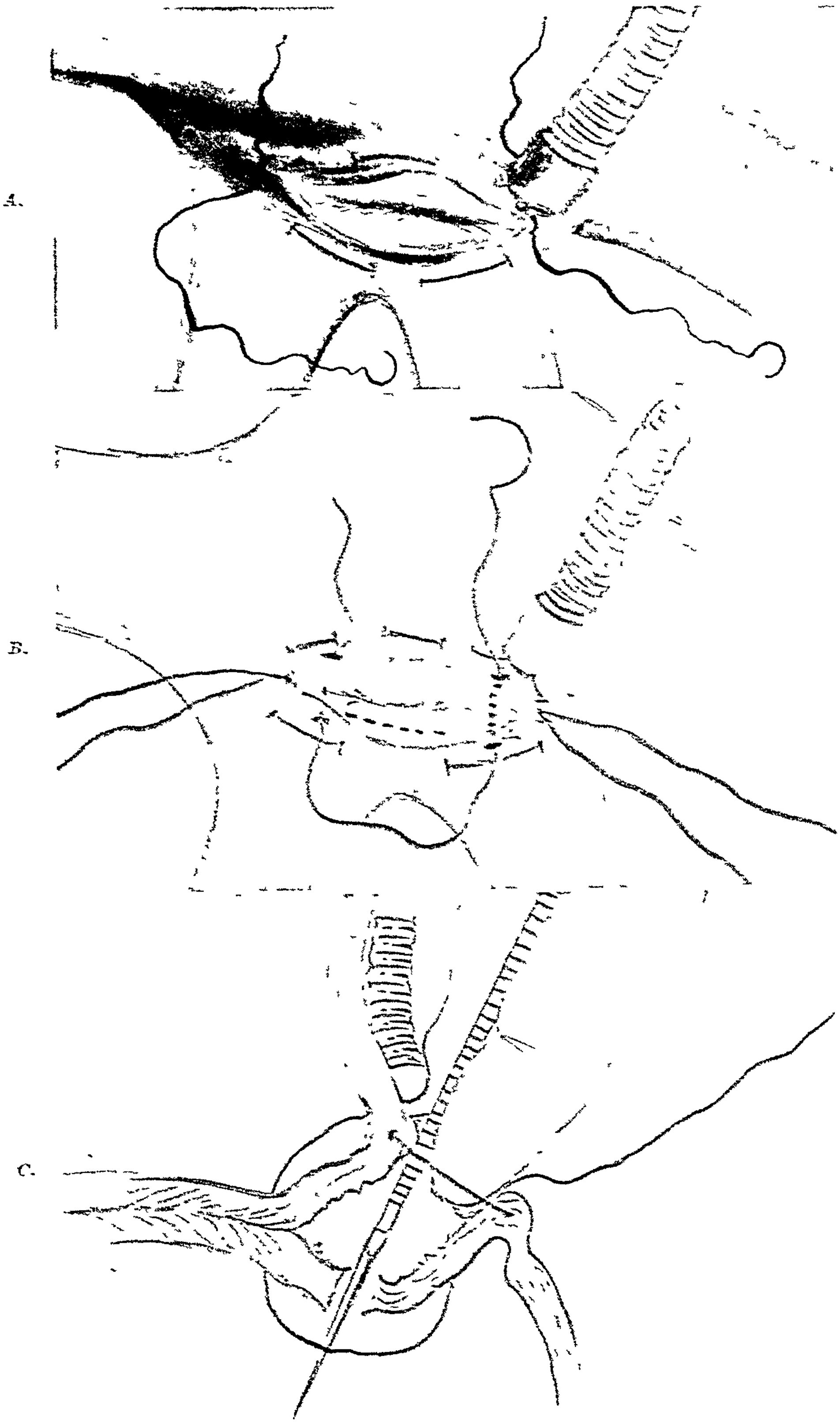


Fig. 15.—A, B, and C. (*For legend see opposite page.*)

appendage in a search for intra-atrial thrombotic masses. If such a clot is found, thereafter it is avoided assiduously in order not to dislodge a fragment.

The finger tip now is inserted deeply into the diminutive valve orifice, by the same movement both measuring the size of the opening and, in favorable cases, bringing about the beginning of commissural separation (splitting). As it passes below the apex of the valve cone, the finger tip attempts to palpate the supporting chordae and papillary muscles to determine their location and condition. In no case, should the finger be permitted to block the valve passageway for a longer time than that occupied by three heartbeats. It must be withdrawn completely into the atrial chamber within this period.

After permitting a few seconds for cardiac recovery, the finger reexamines the valve orifice to determine if any effective enlargement has been produced, if any new regurgitation has been created, and to recognize at which commissure the splitting may have started. Should a beginning split be found, in the absence of an increased amount of regurgitation, disruptive digital pressure then is applied directly at the apex of this area of partial valve divulsion.

Digital Commissurotomy ("Finger Fracture").—Since effective external counterpressure over the commissures seldom is feasible when using this right-sided approach to the mitral valve, a different digital technique is employed from that which is customary with the left sided approach. The apex of the intracardiac finger is placed upon the atrial aspect of the valve at a point just peripheral to one of the extremities of the valve slit. Gradually increasing pressure is applied to the valve margin, being directed toward the apex of the heart. Soon, the bare finger tip will slip over the apex of the valve commissure through the orifice and into the left ventricular chamber. This maneuver momentarily applies considerable but controlled divulsive force to this extremity of the valve without incurring significant risk of injuring the wall of the heart (Fig. 17, *A* and *B*). Basically, it utilizes the principle of the paper knife.

In favorable cases, the fused leaflets will separate a few millimeters with each such stroke, and this may be repeated again and again until the operator is convinced that as complete a separation of this commissure has been brought about as can be accomplished by blunt pressure. When the lengthening of one extremity of the valve slit has been extended fully to the annular ring, which is readily palpable as a circular cordlike structure completely encircling the basal portion of the valve substance, the surgeon's attention must be directed toward the opposite one (Fig. 17, *C*).

Frequently, at one or both commissures, only a limited or no separation may be achieved even by persistent repetition of this maneuver. Generally, this will lead to the conclusion that an instrumental attack upon the valve is indicated.

Creation of Mitral Regurgitation.—In a certain percentage of patients, at some stage in the process of valve splitting, a new regurgitant jet will be detected which, when first noted, will be found to be lo [illegible] th
of the dissection. This jet will be small and of high vel

chamber. During the subsequent maneuvers, care must be taken not to compress the right atrium and venae cavae so firmly as to shut off the flow into the right side of the heart. Even lesser degrees of pressure, if maintained, may bring about severe hypotension, perhaps by a reflex mechanism. When the valve orifice is reached, it is examined digitally. The possible existence of insufficiency is sought for, and if a regurgitant jet is noted, an attempt is made to evaluate its relative size (1 to 4 plus) and to localize it with respect to the position of the extremities of the valve slit. The circumorificial portion of the valve is palpated in an attempt to determine the relative flexibility or rigidity. The presence of any calcification and its amount, localization, and character are noted. In those cases in which the calcific deposition takes the form of loosely attached sandlike particles or even of stalagmites (Fig. 16), special care must be taken not to cause encrustations from breaking off during the required valvular manipulations. The incidence of calcification of the mitral valve as determined by digital examination is noted in Table X.

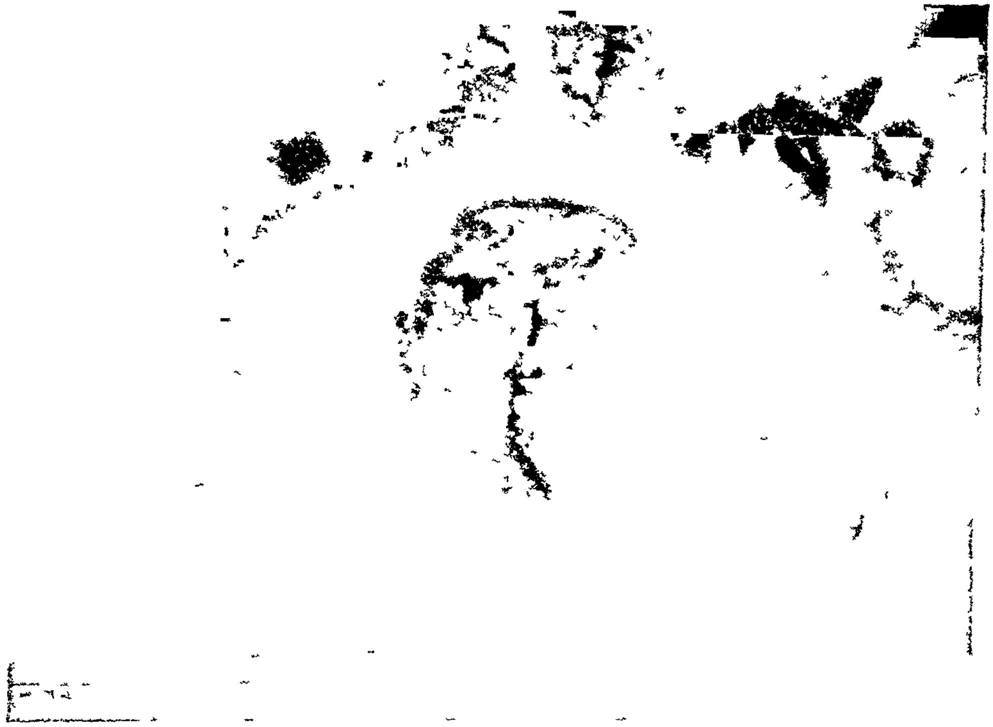


Fig. 16.—Photograph of mitral valve showing loosely attached calcific sand which readily may become detached during the necessary manipulations of the valve.

TABLE X. INCIDENCE OF CALCIFICATION OF THE MITRAL VALVE IN 200 MITRAL COMMISSUROTOMIES FROM THE RIGHT SIDE COMPARED WITH THE FIRST 1,000 CASES FROM THE LEFT

| SIDE | NUMBER OF CASES | NUMBER CALCIFIED | PER CENT |
|---|---|---|---|
| Right | 200 | 85 | 42.5 |
| Left | 1,000 | 370 | 37.0 |

The finger sweeps generally around the lining of the cardiac chamber and explores especially the posterior atrial wall and the region of the left atrial effort might well increase the over-all leak to lethal proportions. Furthermore, full separation of the valve tissue will tend to inhibit or to delay the subsequent development of valvular restenosis which here represents our best hope for "spontaneous" correction of the regurgitation.

OBJECTIVE: BICOMMISSURAL MOBILIZATION.—When both commissures have been separated to the valve ring (annulus fibrosus), the opening is considered anatomically to be as large as can be produced in the given patient. Usually, in the average-sized adult patient, this will be appreciated and designated as a 2 finger opening or larger (estimated 4 to 6.0 sq. cm.) However, with fibrotic contraction or dilatation of the annulus there is corresponding variation in the actual size of the opening which ma h

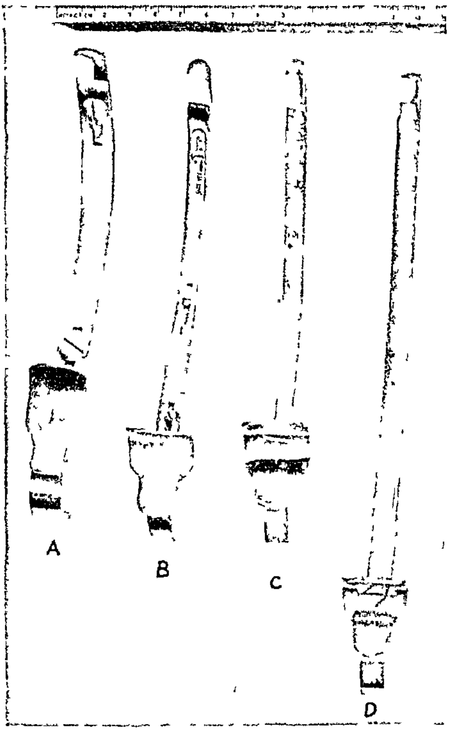


Fig. 18—Progressive modifications of the guillotine knives used in the performance of mitral commissurotomy.

*A*, Original primitive broad-bladed guillotine knife designed in 1949

*B*, Reduction in size and refinement by O'Neill in 1950

*C*, Streamlining of ensheathed guillotine knife using tongue-in-groove principle, by Bailey and Glover in 1951

*D*, Lengthening of instrument for use by the right-sided approach and incorporation of the sharp underjaw in conformity with the papillotome principle of Nichols in 1955 further attempt to achieve additional separation of the valvular tissue at this commissure is almost certain to be attended by a disproportionate increment of increase in the amount of incompetence. Then the regurgitant stream becomes less "jet-like" and more diffuse, involving a greater portion of the valve aperture. It is felt, therefore, that the first recognition of a digitally detectable jet of created regurgitation, occurring at the valve extremity which is being opened, constitutes a contraindication to any further manipulation of this portion of the valve. Its creation probably has been due either to a relatively small deviation of the commissural cleavage from the true anatomic line of rheumatic cusp fusion, or to an extension of the splitting of the valve components beyond the limits of adequate chordal support. However, the appearance of a minor degree of regurgitation at one extremity of the valve should not deter the operator from attempting to separate the opposite commissure. On the other hand, should a major or even a moderate amount of incompetence be created during the surgical mobilization of one commissure, no attempt should be made to open the other one. Such an

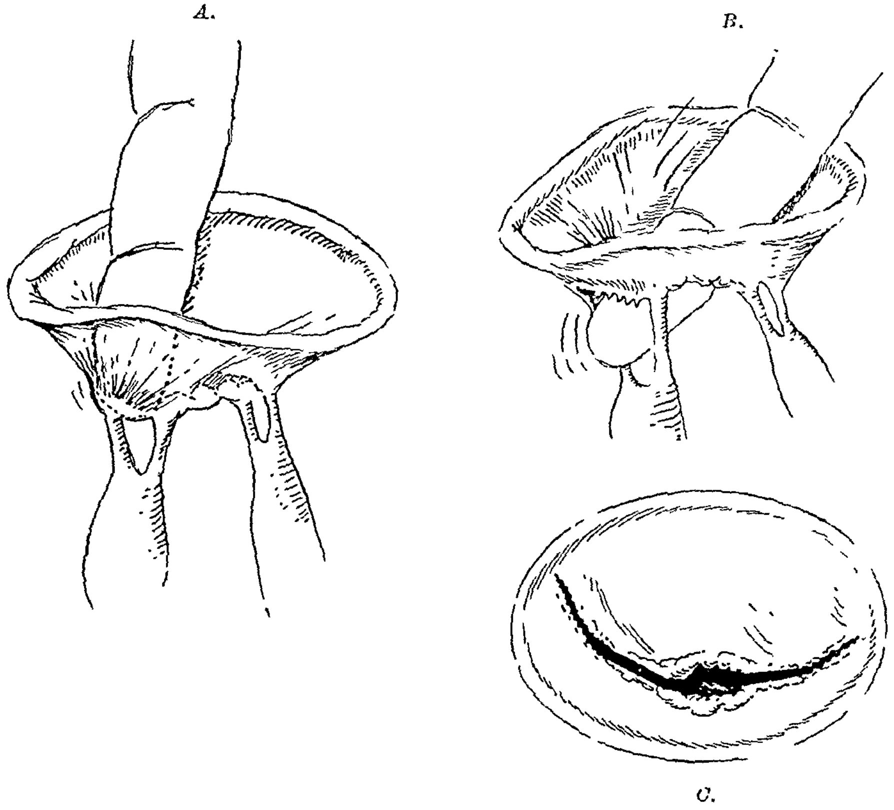


Fig. 17.—Application of divulsive pressure to one of the valve commissures by the right-sided method.

*A*, Pressure is applied by the finger tip against the fused tissue just beyond the actual valve extremity, being directed toward the ventricle.

*B*, As the pressure is increased, the finger tip slips toward the apex of the valve slit and through its orifice, tending powerfully to separate or disrupt the valve tissue along the line of fusion (paper-knife effect).

*C*, Valve separation extended to annular ring. The lines of splitting begin horizontally, but then turn upward at either valve extremity.

Harken and Black[20] have "violently condemned" the use of the guillotine valvulotome as "indelicate and clumsy," and have recommended instead their "bread knife." While our experience with the latter instrument admittedly is limited, there is unquestionably a serious risk of dividing the supporting chordae to one of the leaflets when the end of such an instrument is passed well below the valve level, as is necessary with any valvulotome which is used in a sawing manner (Fig. 19, *A* and *B*). On the other hand, the tapering probe-like underjaw of the modern guillotine facilitates its close application to the leaflet substance without danger to the chordae (Fig. 20). However the question is not whether a surgical instrument has been adapted from the French Revolution or merely borrowed from the kitchen, our point is that many different types of valvulotome may be found efficient and safe in the hands of those who have become skilled in their use.

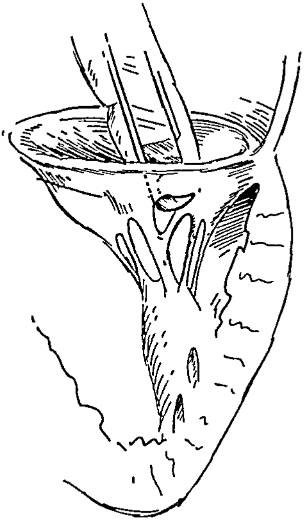


Fig. 20.—In contrast the very narrow pointed underblade of the modern guillotine knife permits its accurate application immediately against the undersurface of the commissural tissue even when there is shortening of the "yoke" of chordopapillary suspension or oblique chordal suspension.

TECHNIQUE OF INSTRUMENTAL COMMISSUROTOMY WHEN USING THE GUILLOTINE KNIVES.—The guillotine may be passed through the valve opening and brought against the apex of the appropriate commissure in the closed state. If this technique is used, it is then advanced sufficiently to extend slightly below the ventricular aspect of the valve substance, and is pressed against the commissure in the line of leaflet fusion. The jaws of the instrument are opened by retracting the sliding blade, thereby perm [illegible] lower [illegible] le

Unfortunately, extreme fibrosis or calcification of the leaflets may render their motion sluggish, so that the restored functional adequacy of the valve may not be proportional to the anatomic extent of leaflet separation.

When it is impossible to mobilize one or both extremities of the valve fully by blunt digital pressure, instrumental division of the fused commissural substance, or at least of the dense marginal tissue, is necessary. While valvulotomes of various types might well be effective, we nearly always have used somewhat elongated versions of our original right and left guillotine knives. These have been modified as suggested by Nichols[12] so that they may be used also to divide an extensive cross-fusion of the subvalvular supporting tissue (Fig. 18, *A, B, C, D*).* They are inserted individually, in the closed state, into the left atrial chamber by passing them along the palmar surface of the bare left index finger as it extends into the cardiac chamber, while the anterior lip of the dissected interatrial groove is held up by tissue forceps to facilitate the passage. These instruments are so streamlined that they fit the finger smoothly without incurring a significant blood loss.

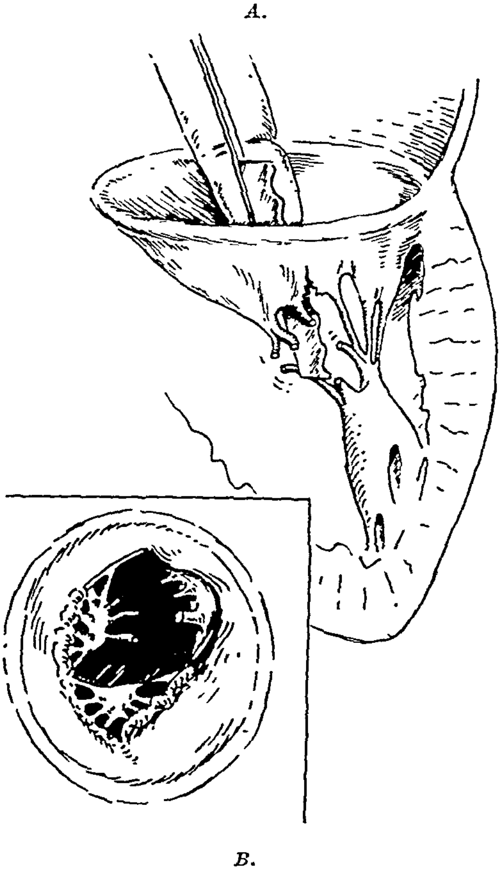


Fig. 19.—*A*, Danger of inadvertent division of the chordae supporting the septal leaflet when the "bread knife" is used to divide a valve commissure. This is an inherent risk associated with the use of any valvulotome so designed that it must be extended deeply below the valve margin, especially when a sawing type of motion is required. This danger is greatest when the papillary muscle has become adherent laterally to the ventricular wall so that the chordae to the septal leaflet run obliquely across (below) the valve orifice. *B*, Insert showing flail-leaflet so produced.

---

*Obtainable from George P. Pilling & Son Co., Philadelphia, Pa.

While the authors frequently have used this method of application of the guillotine, they usually have preferred, after introducing the closed instrument into the left atrial lumen, to open its jaws for a distance of approximately 1 cm before passing the beaked underjaw through the valve orifice and beneath the commissure (Fig 22) Again, it is stressed that extreme care must be taken to ensure that it has been placed between the two parallel columns of supporting chordae and in line with them. It must lie above any cross-fused chordae. The plane of the instrument's first cut will be nearly horizontal in the majority of patients

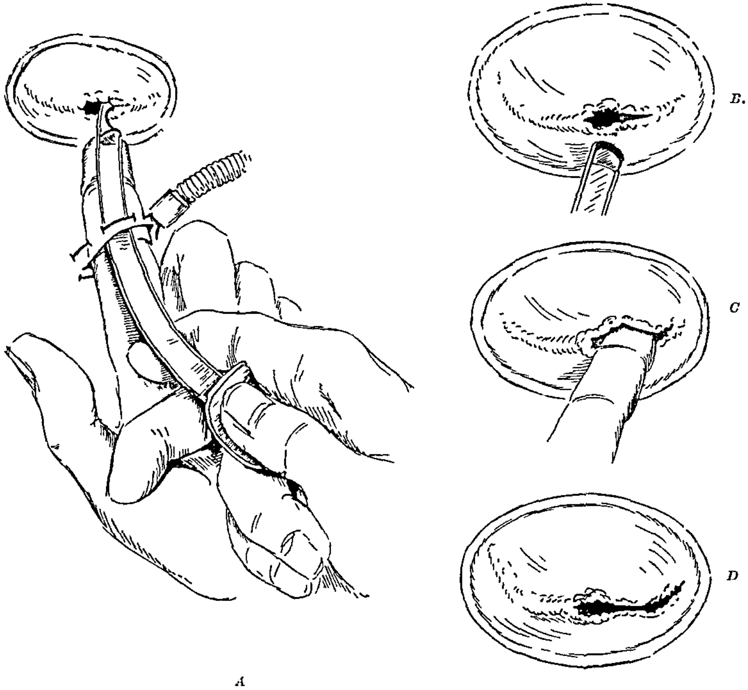


Fig. 23.—*A*, The proper line of direction of instrumental division of the valve will at first lie nearly in the horizontal plane especially at the posteromedial commissure

*B*, Closure of the instrument by advancement of the sliding blade shears through 4 mm of scarred tissue The closed head of the valvulotome then is withdrawn from the valve passage completely into the atrial chamber

*C*, An attempt at extension of the separation by digital pressure now may prove effective since the continuity of the heavily scarred marginal tissue has been interrupted

*D*, If this occurs the line of subsequent splitting usually will curve upward before reaching the annulus fibrosus

The operator's right thumb forces the sliding blade home, the index and middle fingers of that hand providing counterpressure Merto slide beneath the fused commissural tissue. Digital palpation of the undersurface of the valve now establishes that the underjaw of the instrument has been placed accurately in line with and between the two parallel rows of supporting chordae and above any area of possible chordal fusion (Fig. 21, *A*, *B*, *C*).

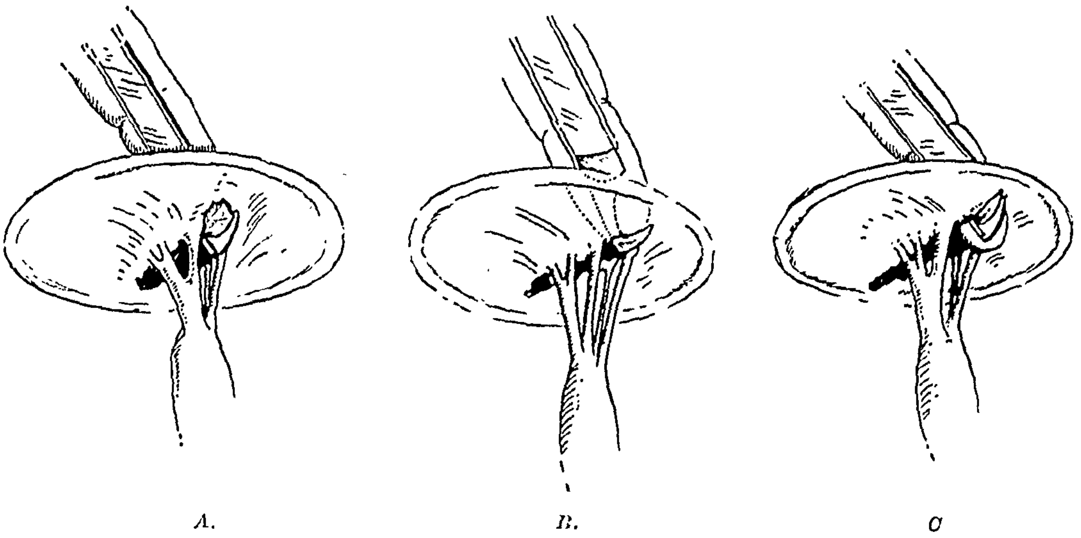


Fig. 21.—Application of the guillotine knife to a fused valve commissure by the closed method.

*A, The appropriate right or left instrument is inserted along the palmar surface of the* intracardiac finger and is advanced in the closed state down to and through the valve orifice. It *is* pressed *firmly* against the apex of the commissure and in line with it.

*B*, As the sliding blade is retracted, the pointed underjaw slides under the commissure. *The finger tip determines that it lies accurately* between the two parallel rows of supporting chordae.

*C, The sliding blade is driven home* by the thumb of the right hand, shearing through 4 mm. of scarred marginal valve tissue.

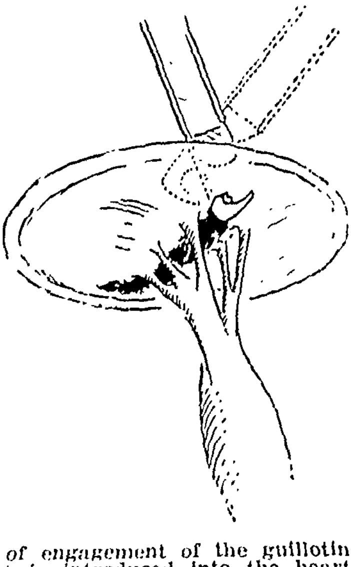


*Fig. 22.—Open technique of engagement* of the guillotine knife against the valve commissure. While the instrument is introduced into the heart in the closed state, the jaws are *separated before* introduction through the valve orifice. By slight angulation, the pointed underblade of the opened instrument may be inserted readily through even the most diminutive orifice and engaged under the fibrosed commissure. Again care is taken that it comes to lie in line with and between the two parallel rows of supporting chordae.

This may be indicated by palpation of a linear elevation or depression upon the atrial surface of the valve as mentioned previously. This marks the line of fibrous juncture of the cusps. Although the two columns of supporting chordae can be felt below the valve, their complete distribution to the leaflets may be difficult to make out until the valve mobilization has been completed. It is felt that, as with the right commissure, the first incision of the fused

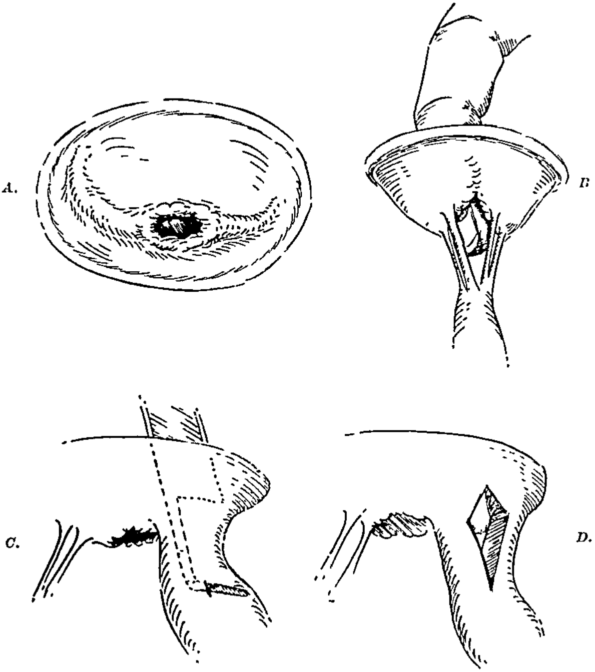


Fig. 24.—*A*, Linear fibrous elevation upon the atrial aspect of the valve which represents the line of leaflet adherence may guide the placement of the valvulotome.

*B*, Recognition by palpation of the two rows of supporting chordae below the valve similarly may orient the surgeon in his placement of the valvulotome.

*C*, If there is no linear elevation upon the atrial aspect of the valve, and if there is only a single massive pillarlike fibromuscular support below the valve, the pointed underblade of the instrument simply may be driven deeply into the middle of this pillar in a horizontal direction.

*D*, Then, closure of the guillotine will start the proper division of both the valve and its support.

anterolateral commissure should be made in a horizontal direction unless a prominent radiating linear elevation or the curve of the valve slit during ventricular systole clearly delineates an immediate upward deflection of the natural line of leaflet separation. A second cut, when necessary, begins at the apex of the first and may be permitted to deviate [illegible] the surface of the curved portion of the handle of the fixed blade (Fig. 23, *A*). The upper blade of the instrument crosses the lower, shearing through 4 mm. of scarred or calcified tissue. *With the jaws closed*, the lower end of the knife is withdrawn into the atrium and the heart is allowed time to recover from any disturbance incident to the manipulation. We frequently prefer to remove the instrument completely from the heart at this time. However, this will necessitate reinsertion if further cutting should be required. As soon as the anesthetist reports that the level of the blood pressure and the strength of the pulse have been maintained at (or restored to) reasonable levels, the finger tip is advanced to examine the valve with respect to the extent of commissural division and especially to detect any insufficiency which may have been created. Should a new regurgitant jet be found, it nearly always will be minimal, but its very existence should suffice to contraindicate any further manipulation or instrumentation of this portion of the valve mechanism.

Usually, no regurgitation will have been produced, and a further effort at digital splitting of the valve may prove effective now that the continuity of the tough margin of the value orifice has been interrupted (Fig. 23, *B, C*). If a new attempt at digital splitting fails, further instrumentation is indicated until the valve opening extends to the annulus fibrosus, or until the first sign of beginning insufficiency is noted. Should digital pressure become effective at any time (tested by tentative pressure after each episode of instrumentation), it promptly should be substituted for the use of the valvulotome. The line of valve splitting will then normally curve upward (Fig. 23, *D*).

THE POSTEROMEDIAL COMMISSURE.—Not only will the operator find that the posteromedial commissure may be opened readily by the right-sided approach, but it also will be evident that it more appropriately might be designated as the "right" commissure since at this extremity the diminutive valve slit is directed nearly horizontally (then slightly upward) and toward the right. Frequently, the exact line of inflammatory valve fusion will be indicated by a palpable linear elevation or depression upon the atrial surface of the valve which runs from the commissures toward the annulus fibrosus (Fig. 24, *A*). An even more reliable and constant guide to the line of an obliterated commissure is provided by the two parallel rows of chordae which individually *support this extremity* of each of the respective leaflets. In those relatively few valves in which the subvalvular supports have become agglomerated into massive pillars of fibromuscular tissue (Fig. 24, *B, C, D*) and where the pathologic abnormalities of the atrial surface of the valve obliterate the usual landmarks, experience alone can guide the surgeon in selecting the proper line of division. It is well to remember that the proper line for beginning separation of the right commissure runs essentially in the horizontal plane when the patient lies in the supine position (as stated previously).

THE ANTEROLATERAL COMMISSURE.—It is slightly more difficult to place the anterolateral commissural separation anatomically since the line of fusion (leaflet closure) usually starts horizontally, but soon curves sharply upward.

natural curve of valve approximation. After division of the tough free valve margin anterolaterally, further separation of the tissues often may be accomplished *by digital pressure alone.* This blunt *splitting tends to follow the* natural anatomic line of valve fusion (Fig. 25 *A D*).

Further instrumentation of this extremity of the valve should be continued until digital splitting becomes possible, until a fully adequate valve opening is achieved, until the line of leaflet separation reaches the annulus fibrosus, or until the first sign of created regurgitation at this pole of the orifice interdicts further efforts. In general, our attempt should be to extend the leaflet mobilization at both extremities fully and naturally to the annulus fibrosus (clearly detectable to palpation), but never beyond this limit.

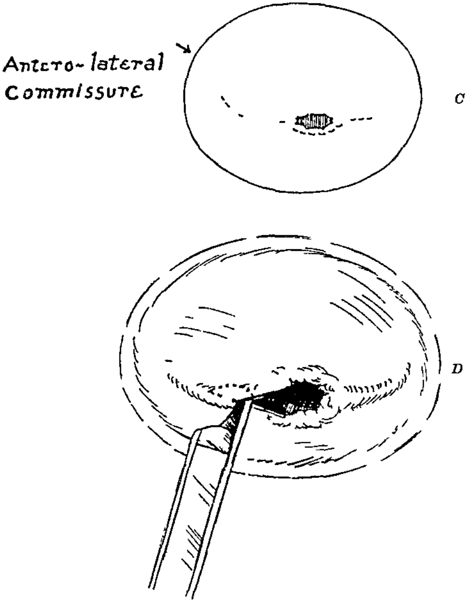
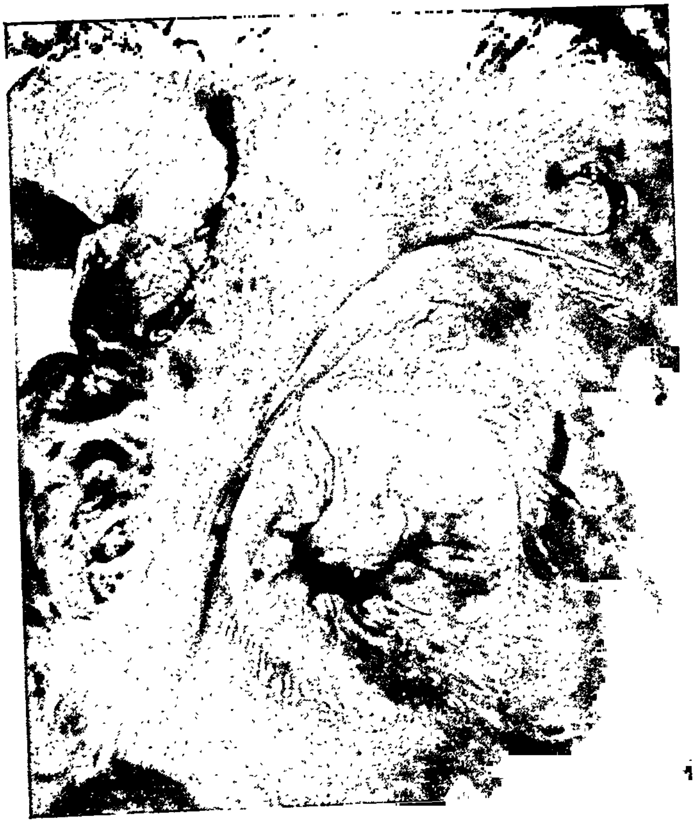


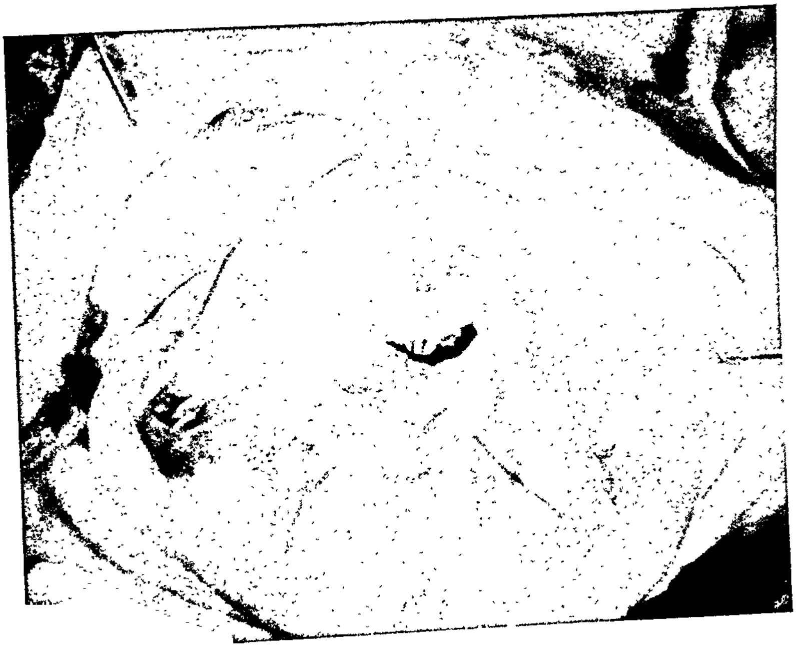
Fig. 25 Cont'd.—*C*, Average line of curvature which the instrumentalist may follow with reasonable confidence in the absence of any more definite guide.

*D*, Application of open guillotine against the apex of the previous cut for a second one. The first section may have been made horizontally. The subsequent ones should be caused to deviate somewhat in an upward curve.

Table XI indicates the variations in technique of commissural separation which were used in our series. It is understood that in many of the valves the *mobilization was begun instrumentally, as indicated, and then was completed* by digital pressure (not indicated in the table).



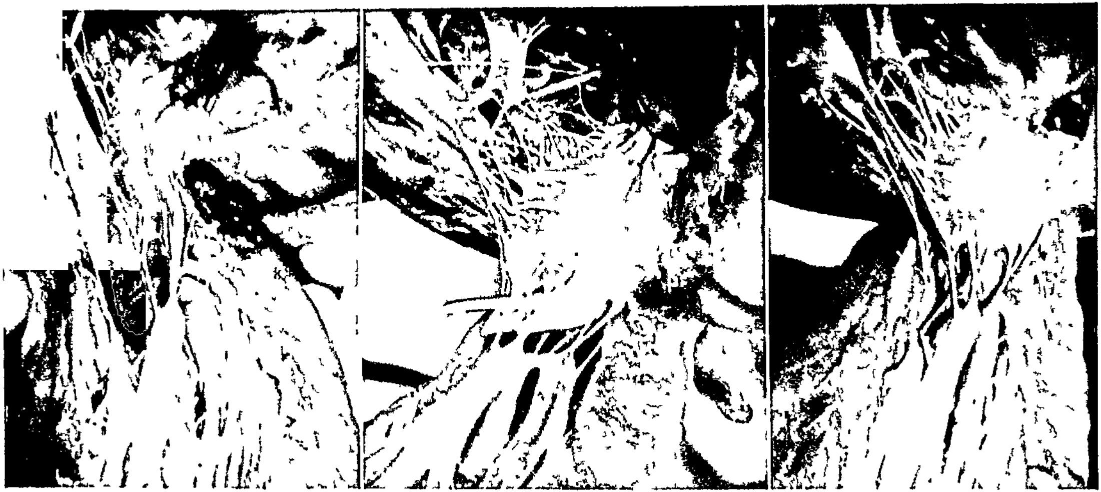
*A.*



*B.*

Fig. 25.—*A*, Photograph of stenotic mitral valve showing linear depression corresponding to a markedly curved line of valve fusion. (Courtesy Dr. John Storer and New York J. Med., March 15, 1956.)

*B*, Photograph of stenotic mitral valve showing a lesser arc of curvature which results from a different type of valvular distortion. (Courtesy A.M.A. Book of Exhibits, 1955.)



*C* *D 1* *D 2*

Fig. 3 Cont'd.—*C*, Such a papillary split may be extended by finger tip pressure perhaps to its ventricular attachment. *D 1* and *2*, In resistant cases definitive longitudinal division of the papillary mass may be accomplished by boldly piercing it with the sharp pointed underbeak of the papillotome or modified guillotine knife and then closing the instrument. (Photograph.)

TABLE XI. DIGITAL OR INSTRUMENTAL OPENING OF COMMISSURES IN 200 MITRAL COMMISSUROTOMIES FROM THE RIGHT SIDE

| | NUMBER OF CASES |
|---|---|
| Both commissures opened digitally | 28 |
| Anterior commissure opened instrumentally, and posterior digitally | 26 |
| Posterior commissure opened instrumentally, and anterior digitally | 25 |
| Both commissures opened instrumentally | 116 |
| Anterior commissure opened digitally, posterior not separated | 2 |
| Anterior commissure opened instrumentally, posterior not separated | 1 |
| Posterior commissure opened digitally, anterior not opened | 1 |
| Posterior commissure opened instrumentally, anterior not opened | 1 |
| Total | 200 |

SUBVALVULAR STENOSIS.—The chordae supporting the respective leaflets were found to be mutually cross-fused in such a way as to reinforce seriously the inflammatory union of the two thickened leaflets in 29 of our first 200 patients with stenotic mitral valves operated on from the right side (Table XII). In some individuals, one or both of the papillary muscles were found to be attached directly to the valve apex (see Fig. 5, *C*). In either instance, after recognition of the condition, the first effort was a cautious but determined effort to separate the leaflets themselves. After the leaflets had been mobilized anatomically an attempt was made by direct downward (toward the apex) pressure of the tip of the bare index finger to separate any remaining fused chordopapillary structures (Fig. 26, *A*).

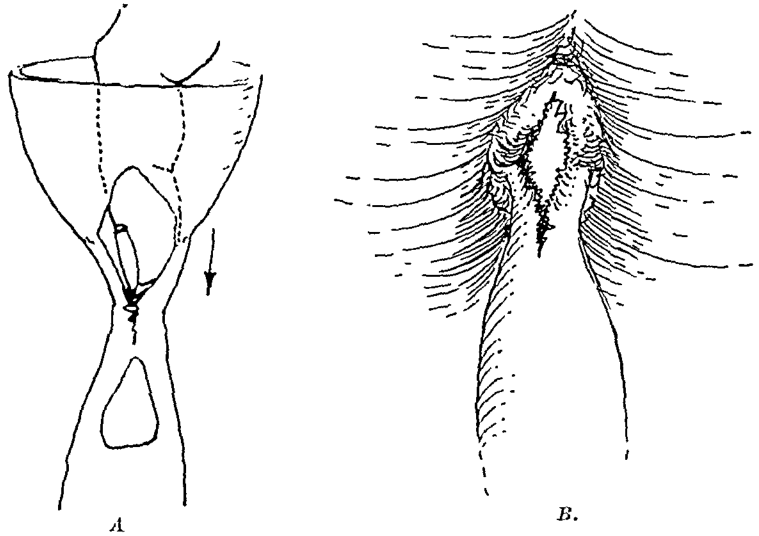


Fig. 26.—Relief of subvalvular obstruction. *A*, Separation of cross-fused chordae by direct end-on pressure with a finger tip. Often this may be accomplished inadvertently during digital mobilization of the valve commissure, perhaps even without recognition of the condition.

*B*, A beginning split of a fused papillary mass may be produced during the separation of the commissures.

This program is especially effective against lightly cross-adherent chordae. If the leaflet separation inadvertently (as is usual) has established a beginning division or laceration of the "top" of a complex papillary fusion mass, it may be possible easily to extend this beginning longitudinal "splitting" of the

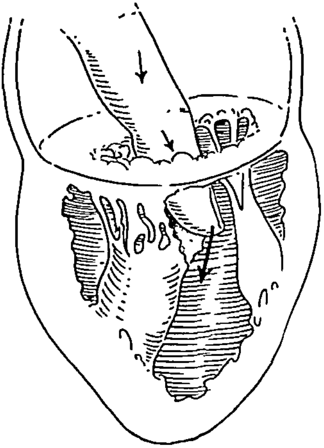


Fig 27.—Occasionally only an eccentric passageway can be established through a badly distorted valve. However, if the over-all size is adequate, the clinical result usually is good (Bailey: Surgery of the Heart, courtesy Lea & Febiger, p 587 )

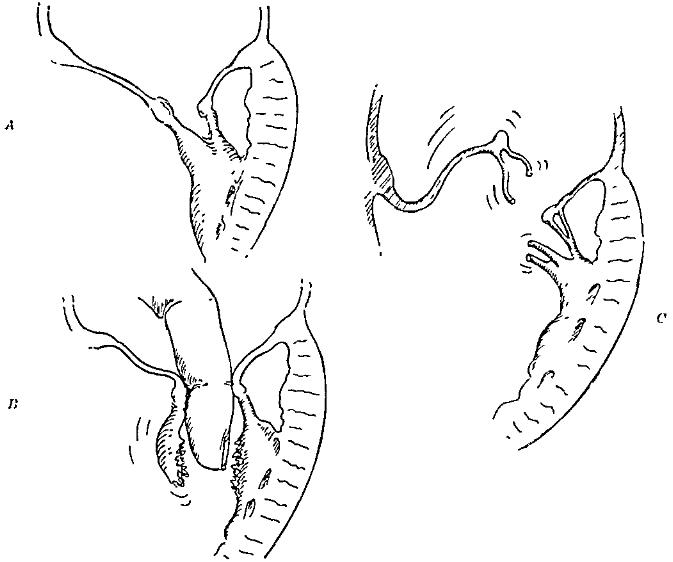


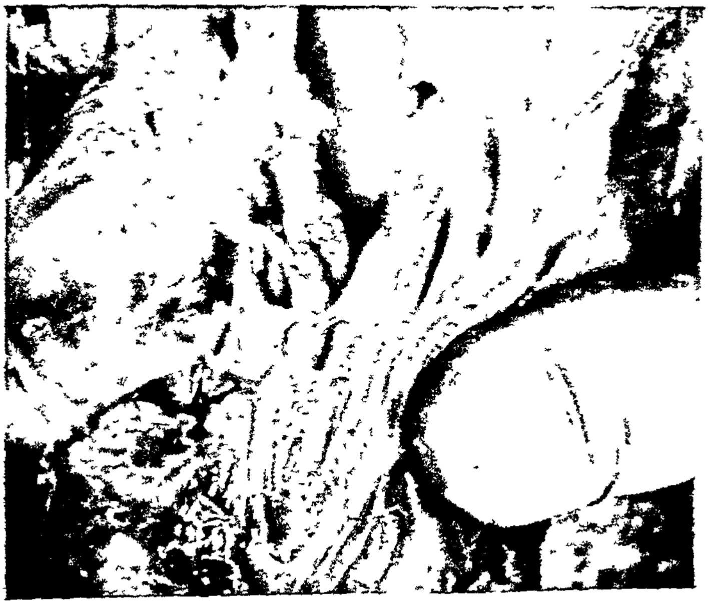
Fig 28.—*A*, In some instances, extreme lateral fusion may have taken place between a papillary muscle and the ventricular wall. This may be suspected from the course of the chordae which support the septal leaflet

*B*, Should splitting of such a papillary muscle prove necessary, great care must be taken to so direct the line of created separation anatomically lest one side of the split muscle may become entirely detached with ensuing loss of valvular support and consequent severe regurgitation

*C*, In no instance should any such obliquely or transversely directed chordae be sectioned lest a flail valve be produced.

TABLE XII. DISTRIBUTION OF SUBVALVULAR STENOSIS AS RECOGNIZED IN THE FIRST 200 CONSECUTIVE MITRAL COMMISSUROTOMIES PERFORMED FROM THE RIGHT SIDE

| | | NUMBER OF CASES |
|---|---|---|
| *Location* | | |
| Anterior (chordae only) | | 9 |
| Posterior (chordae only) | | 4 |
| Anterior and posterior (chordae only) | | 6 |
| General fusion and shortening of chordae and papillary muscles (pillarlike masses) | | 10 |
| | Total | 29 |
| *Treatment* | | |
| Not treated separately—split with leaflet mobilization | | 12 |
| *Treated* | | 17 |
| Split digitally (independently) | 11 | |
| Dense papillary adherence | | |
| Cut with guillotine | 4 | |
| Cut with papillotome | 2 | |
| | Total | 29 (14.5%) |



E.

Fig. 26. Cont'd.—E. Digital splitting of the longitudinally partially divided papillary mass then becomes possible.

papillary muscle mass simply by end-on pressure with the finger tip (Fig. 26, *B*, *C*). In more resistant cases, Nichols'[32] technique of instrumentally starting the separation of the muscle mass may be necessary as it was in 6 of our first 200 patients (Table XII; Fig. 26, *D*, *E*). In some individuals, a subvalvular fusion mass may prove inseparable at one valve extremity, while no subvalvular obstruction may exist at the opposite one, or may readily be overcome. Such resulting eccentric or diagonal entrance passages probably hamper free atrioventricular blood flow to some extent, but in our experience the clinical end result usually has been entirely satisfactory (Fig. 27).

of the groove upon the completion of the dissection. After this first suture has been run the length of the cardiac wound, it is tied to the short end of the similar stitch which has been placed at the opposite extremity. The needle-bearing end of this latter suture is used to reinforce the placed mattress layer in an over-and-over running fashion Its lead end, in turn, is tied to the short end of the first (Fig. 30, *A*, *B*) As the lips of dissection are approximated, the tourniquet tension upon the purse string is released to avoid puckering

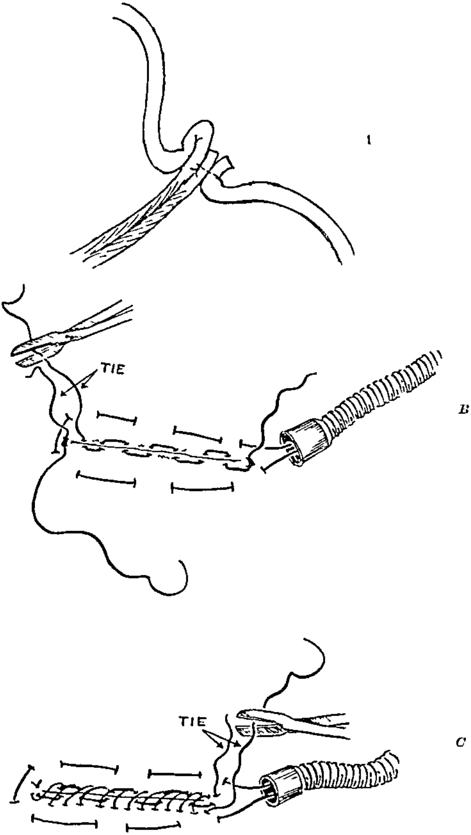


Fig 30 —Closure of the cardiac wound

*A*, Tying down the deep nylon mattress suture everts the lips of the left atrial incision

*B*, One of the stay sutures at the lower extremity of the dissection is used to place a continuous mattress suture line which approximates the two lips of the cardiac wound The leading end of this suture is tied to the short end of the opposite one

*C*, The long (needle-bearing) end of this second stay suture is then used to place a row of continuous running stitches just superficial to the mattress layer. It in turn, is tied to the short end of the first stay suture The purse string is removed completely, if possible

Normally, both papillary muscles arise from and are attached to the mural (lateral) wall of the left ventricle, fairly well down toward the cardiac apex. In some of these rheumatic patients, a variable amount of lateral adherence between these muscles and the endocardial surface of the lateral wall of the ventricle will have taken place, so that the muscles appear to have a higher (toward the atrium) attachment and to be directed more obliquely with respect to the atrioventricular channel than is normal. This morbid anatomic possibility must be taken into consideration when attempting to split digitally or to divide instrumentally such a muscle mass in its longitudinal axis (Fig. 28, *A*, *B*).

Furthermore, it is essential to realize that when one or both papillary muscles are directed thus obliquely toward the valve orifice, the corresponding chordae which attach to the septal leaflet will be bound to "run across" the valve passageway. Since they support this leaflet, they must not be divided lest severe regurgitation be created (Fig. 28, *C*). In these cases, any long cutting type of instrument (such as the "bread knife") which must be passed deeply below the valve level may be dangerous (see Fig. 19).

CLOSURE OF THE DISSECTED INTERATRIAL GROOVE.—After the definitive valvular surgery has been completed either to the surgeon's complete satisfaction or at the level of the best compromise possible under the existing circumstances, the intracardiac operating finger is withdrawn cautiously as the purse-string suture is tightened progressively. If there is any persistent bleeding, the two lips of the dissected interatrial groove may be approximated temporarily for hemostasis with a pair of Nelson tissue forceps (Fig. 29).

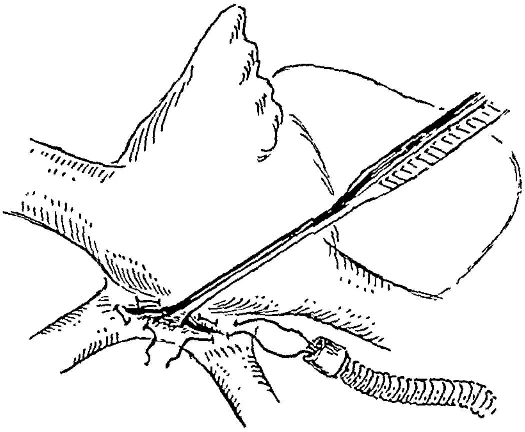


Fig. 29.—Method of control of bleeding between the lips of the dissected interatrial groove. This simple measure may prove lifesaving should the purse-string suture become divided.

This simple measure may prove lifesaving in the event of inadvertent section of the encircling hemostatic purse-string suture. The ends of the previously placed nylon mattress suture now are tied down, bringing about eversion of the lips of the left atrial incision and assuring accurate endocardial apposition within the atrial chamber.

The two lips of the dissected groove are reapproximated with a continuous horizontal everting mattress suture, using one of the No. 3-0 cotton stay sutures with swedged-on curved needles which were placed at each end

The over-all operative mortality in the entire 200 patients (Table XIV) with all concomitantly performed procedures was 11 per cent (22). One was an additional late death in a patient in whom aortic commissurotomy was performed simultaneously, due to the late development (six months) of subacute bacterial endocarditis.

TABLE XIV MORTALITY (EARLY AND LATE) IN 200 OPERATIONS FOR ISOLATED AND COMPLICATED MITRAL STENOSIS USING THE RIGHT SIDED APPROACH

| | NUMBER OF CASES | NUMBER OF DEATHS |
|---|---|---|
| *Complicated Mitral Stenosis* | | |
| Aortic commissurotomy | 28 | 4 |
| Aortic annulus constriction for insufficiency | 2 | 1 |
| Tricuspid commissurotomy (175 explored) | | |
| For major stenosis | 19 | 5 |
| For minor commissural fusion | 13 | - |
| Tricuspid annulus plication for insufficiency | 2 | 1 |
| Atrio septo pexy (for congenital defect) | 2 | - |
| Atrio septo pexy with surgical transposition of anomalous pulmonary veins | 1 | - |
| Right middle lobe resection for arteriovenous fistula | 1 | - |
| Mitral commissurotomy in the presence of severe mitral insufficiency (not treated at this same operation) | 2 | - |
| *Isolated Mitral Stenosis* | | |
| Mitral commissurotomy for pure or predominant mitral stenosis and insufficiency, *not excluding minor mitral insufficiency* or tricuspid insufficiency | 130 | 11 |
| Total | 200 | 22 |

Three of the 4 patients in whom definitive trivalvular surgery was carried out died. In each, the death was attributed chiefly to the extremely poor condition of the individual.

## EARLY FOLLOW-UP ON SURVIVING PATIENTS

The early postoperative results in this group of cases have been uniformly excellent, although it must be remembered that the longest period since operation is sixteen months. A personal checkup or a physician's report has been obtained in each patient within the past two months. A preliminary survey of 120 patients surviving operation for mitral stenosis pure or predominant (with insignificant mitral insufficiency) through the right-sided approach revealed only one late death due to massive pulmonary embolism (Patient, S. S.). One other case has been unimproved following surgery (Patient, M. F.). This we feel is related to *rupture of chordae tendineae and* the production of mitral insufficiency during an attempt to palpate the aortic valve by way of the mitral orifice.

Diaphragmatic paralysis on the right side, apparently due to injury to the phrenic nerve, has handicapped another patient in attaining a complete recovery (Patient, H. S.). Seven patients have experienced one or more episodes of indeterminate malaise, usually appearing four weeks to eight months postoperatively and associated with fever and chest pain, sometimes with detectable pleural effusion. Conservative treatment with acetyl[illegible] acid (aspirin) or adrenal corticoid substance ([illegible]) has [illegible] most instances. Thoracentesis has been necessa of the inner surface of the left atrial wall. We prefer to remove the purse-string suture completely, but should bleeding recur when the purse string is released tentatively, it may be tied or additional sutures may be placed.

TRICUSPID EXPLORATION.—Tricuspid exploration by passage of the left index finger through an incision made in the right auricular appendage is carried out routinely *following the mitral commissurotomy*. This order of intervention enables the surgeon to be reassured by a second check from another chamber that an atrial septal defect has not been overlooked or created inadvertently during the dissection of the interatrial septum, and permits an immediate repair by the technique of *atrio-septo-pexy*[41] *should one be found.*

COMPLETION OF THE OPERATION.—*Any necessary tricuspid or aortic surgery* for a concomitant lesion having been completed, the pericardium is closed partially with interrupted sutures. This repair must be sufficiently complete to prevent cardiac herniation, but loose enough to permit free drainage of blood and exudate into the pleural cavity. The chest wall is repaired in layers after careful attention has been given to hemostasis. The pleural cavity is drained through a stab wound made in the right seventh intercostal space in the posterior axillary line by a No. 26 Fr. urethral catheter with additional windows *cut into it. It is connected to a simple waterseal drainage* system.

## OPERATIVE MORTALITY

Among the first 130 patients (Table XIII) who were operated on by the *right-sided approach essentially for isolated mitral stenosis (with or without* associated insignificant mitral insufficiency), there were ten hospital deaths (8 per cent). This was almost identical to that experienced with our first 1,200 patients who were operated on from the left side. The clinical impression of our medical associates has been that an appreciably greater percentage of far-advanced cases has been included in the right-sided group. Eight of these deaths occurred among the first 70 operations while we were relatively inexperienced in the technical aspects of this then new and unusual approach. There have been only two deaths among the last 60 patients. There has been only one late death in this series (Patient S. S.); this was due to massive pulmonary embolization in an asthenic, elderly (58-year-old) patient with extremely advanced renal disease.

TABLE XIII. CAUSES OF OPERATIVE AND LATE DEATH IN 11 MITRAL CASES OPERATED ON FROM THE RIGHT SIDE IN 130 CASES OF PREDOMINANT MITRAL STENOSIS WITHOUT SIGNIFICANT REGURGITATION, TRICUSPID, OR AORTIC DISEASE

| HOSPITAL DEATHS | NUMBER OF CASES |
|---|---|
| Mitral insufficiency created or increased | 5 |
| *Systemic arterial embolism* | |
| Operative (calcific) | 1 |
| Postoperative (thrombotic) | 2 |
| *Massive hemorrhage* | |
| Technical accident (purse string tore out) | 1 |
| Unknown (unstable circulation—hypotension) | 1 |
| Total | 10 (8%) |
| Late pulmonary embolization | 1 |

TABLE XV MURMURS AUDIBLE 3 MONTHS POSTOPERATIVELY IN 72 CASES OF PREDOMINANT MITRAL STENOSIS OPERATED ON FROM THE RIGHT SIDE

| | NUMBER OF CASES | PER CENT |
|---|---|---|
| *Diastolic* | | |
| None | 13 | 60 |
| Present { I - 26<br>II - 3<br>III - 0<br>IV - 0 } | 29 | 40 |
| *Systolic* | | |
| None | 31 | 43 |
| Present { I - 15<br>II - 23<br>III - 3<br>IV - 0 } | 41 | 57 |

Note The above Roman numerals refer to the grade of intensity of the m[illegible]r and not to classification of the cases

appearance of the murmur and the production of digitally det[illegible] mitral regurgitation at the time of surgery The murmur was not [illegible] with any compromise in the generally excellent clinical respons[illegible]

While the postoperative period in many of these [illegible] is very short, a dramatic clinical improvement has been evidenced [illegible] in the great majority One hundred and four of the 111 attendin[illegible] sicians contacted have been most enthusiastic in their reports The m[illegible]ity of the patients have returned to greatly increased activity and man[illegible] to full-time work

## SUMMARY

As a result of the rare necessity for the performance of right thoracic exploration in patients with severe mitral stenosis, a simple and satisfactory method has been devised for digital and instrumental entrance into the left atrium from the right side Increasing familiarity with and improvement in the technique of performing mitral commissurotomy from the right have led to its adoption as our routine operation for most patients with this condition

Our experience in the first 200 patients so operated upon clearly indicates that the right-sided approach, in contrast to the more usual left-sided one, nearly always permits the accomplishment of a superior type of mobilization of the fused valve structures In this series, both commissures were divided in 97.5 per cent of the patients At the same time it was found distinctly easier to obtain definitive separation of cross-fused subvalvular supporting tissues when present.

The right sided approach lends itself particularly well to the simultaneous performance of multivalvular surgery in those patients with associated rheumatic disease of the aortic or tricuspid valve Indeed the ease of tricuspid exploration is so great that this maneuver has been carried out as a routine at the conclusion of the mitral operation A surprising number of "silent" but severe stenoses of the tricuspid valve are [illegible] the[illegible] and then may be corrected promptly.

Auscultatory Changes.—Seventy-two of the patients who were operated on for mitral stenosis only were available for personal re-examination by the members of the cardiological staff of our Clinic, three months or more after their surgery. A diastolic murmur at the apex, present in all patients preoperatively, could be heard in only 40 per cent (Table XV). This is in extreme contrast to the general experience following mitral commissurotomy performed from the left side in which the diastolic murmur is abolished in only a minority of instances. Its persistence in 40 per cent of our patients was felt, for the most part, to be related to turbulence of the increased blood flow over rough valve surfaces rather than to any residual obstruction. These impressions were borne out further by comparison of the pre- and postoperative measurement of the diastolic gradient across the mitral valve (Fig. 31, *A*, *B*.). Left heart catheterization or combined left and right heart catheterization was carried out in over one half of these patients and is being reported elsewhere.[44]

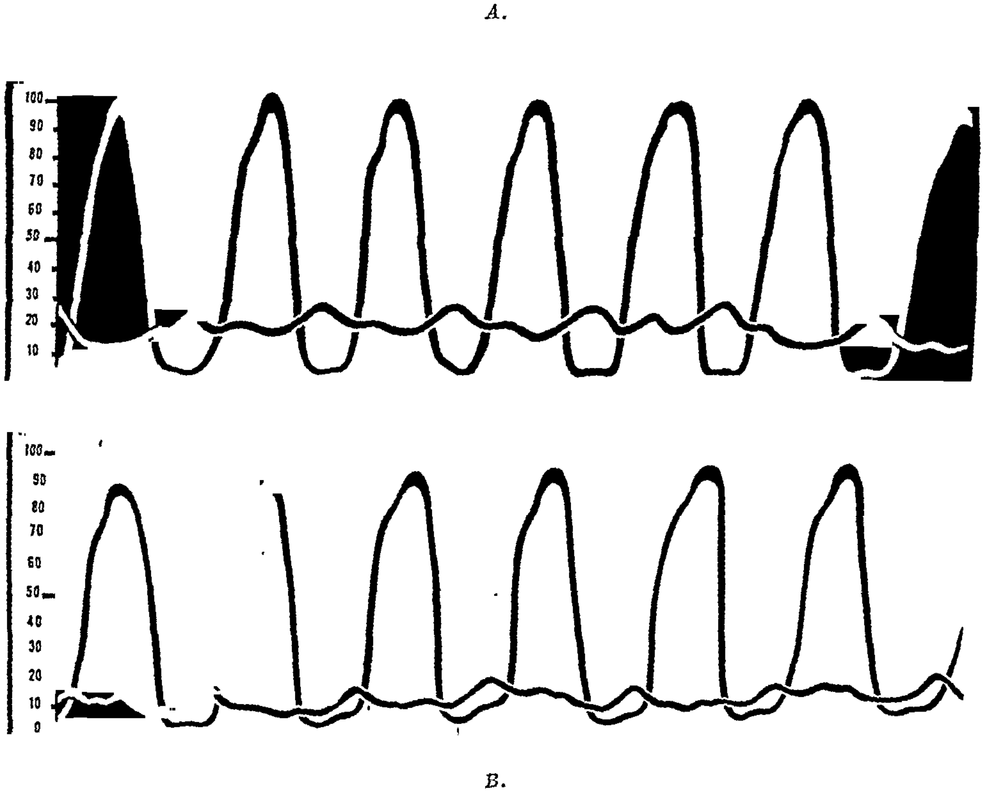


Fig. 31.—Reduction of mitral diastolic pressure gradient as a result of commissurotomy. Left heart catheterization in Patient A. S. (Courtesy of A.M.A. Book of Exhibits, 1955).

*A*, Note the typical abnormal elevation of the gradient which exists during the period of ventricular filling, indicating mitral stenosis.

*B*, Marked reduction in the gradient in this patient following adequate mitral commissurotomy.

A less reassuring finding has been the appearance of a systolic murmur in many patients who did not present one prior to surgery. Such a murmur existed in only 25 per cent, preoperatively, but was noted in 57 per cent, three months later (Table XV). There was no correlation between the

3 Samways, D W Cardiac Peristalsis, Its Nature and Effects, Lancet 1 927, 1898

4 Brunton, L Possibility of Treating Mitral Stenosis by Surgical Methods, Lancet 1 352, and 547, 1902

5 Cutler, E C, Levine, S A, and Beck, C S Surgical Treatment of Mitral Stenosis, Arch Surg 9 689, 1924

6 Smithy, H G An Approach to the Surgical Treatment of Valvular Diseases of the Heart Sixteenth Annual Assembly of the Southeastern Surgical Conference, Hollywood, Fla, April 5 8, 1948

7 Souttar, H S Surgical Treatment of Mitral Stenosis, Brit M J 2 603, 1925

8 Bailey, C P Surgical Treatment of Mitral Stenosis, Dis Chest 15 377, 1949

9 Brock, R C, Baker, C, and Campbell, M Valvulotomy for Mitral Stenosis, Report of Six Successful Cases, Brit M J 1 1283, 1950

10 Harken, D E, Ellis, H B, Ware, P F, and Norman, L R Surgical Treatment of Mitral Stenosis I Valvuloplasty, New England J M 239 801, 1948

11 Brock, R C, Baker, C, Campbell, M and Wood, P Valvotomy for Mitral Stenosis—A Further Report on 100 Cases, Brit M J 47 67, 1952

12 Nichols, H T, and Jamison, W L Subvalvular Mitral Stenosis—Its Recognition and Treatment, J Thoracic Surg 29 611, 1955

13 Husfeldt, E, Copenhagen, Denmark Personal communication

14 Glover, R P, O'Neill, T J L, and Janton O H Analysis of Fifty Patients Treated by Mitral Commissurotomy Five or More Years Ago, J Thoracic Surg 30 436, 1955

15 Ellis, L B, and Harken D E The Clinical Results in the First 500 Patients With Mitral Stenosis Undergoing Valvuloplasty Circulation 11 637, 1955

16 Bailey, C P Surgery of the Heart, Philadelphia, 1955, Lea & Febiger

17 Brock, R C Surgical and Pathological Anatomy of the Mitral Valve, Brit Heart J 14 489, 1952

18 Jordan, P, Jr, and Hellems, H K Mitral Valve Surgery, Surg, Gynec & Obst 95 689, 1952

19 Keyes, J W, and Lam C R Recurrence of Mitral Stenosis Following Mitral Commissurotomy, J A M A 155 247, 1954

20 Harken, D E, and Black, H Improved Valvuloplasty for Mitral Stenosis—With a Discussion of Multivalvular Disease, New England J M 253 669 1955

21 Glover, R P, McDowell, D E, O'Neill, T J E, and Janton, O H Does Mitral Stenosis Recur After Commissurotomy? Circulation 11 14, 1955

22 Harken, D E Personal communication, May 22, 1954

23 Magarey, F R On the Mode of Thickening of the Mitral Valve, J Path & Bact 61 203, 1949

24 Soloff, L A and Zatuchni, J Some Difficulties in Evaluating Functional Results After Mitral Commissurotomy, J A M A 159 673, 1954

25 Soloff, L A, Zatuchni J, Janton, O H, O'Neill T J E, and Glover, R P Reactivation of Rheumatic Fever Following Mitral Commissurotomy, Circulation 8 481, 1953

26 Orr, L M Discussion of J F McCarthy A Critique of Present Methods in the Surgery of the Prostate Gland J Urol 45 432, 1941

27 Letters of Horace Walpole, 1750 1756, Collection of Mrs Paget Toynbee, Letter 382, Page 204

28 Neptune, W B, and Bailey, C P Mitral Commissurotomy Through the Right Thoracic Approach, J Thoracic Surg 28 15, 1954

29 Harken, D E Discussion at Meeting of the Post Graduate Course in Cardiovascular Disease of the American College of Physicians, Boston, Mass Dec 1, 1955

30 Nichols, H T Mitral Insufficiency Treatment by Polar Cross Fusion of the Mitral Annulus Fibrosus, J Thoracic Surg 33 102, 1956

31 Bailey, C P, Bolton, H E, Jamison, W L, and Nichols, H T Commissurotomy for Aortic Stenosis Circulation 9 22, 1954

32 Glenn, W W L The Digital Exploration of the Ventricular Chambers of the Heart Through a Rubber Diverticulum, Yale J Biol Med 25 233, 1953

33 Swann, W K, Bradsher, J T, Jr, and Rodriguez Arroyo, J Intercardiac Surgery With the Aid of Artificial Operative Tunnels, J Thoracic Surg 28 266, 1954

34 Bailey, C P, and Bolton, H E Surgical Treatment of Aortic Stenosis, Cardiovascular Surgery, Philadelphia, 1955, W B Saunders Company, p 272

35 Reale, A Goldberg, H Denton, C, and Likoff, W Rheumatic Tricuspid Stenosis A Clinical and Physiological Study With a Suggestion Method of Diagnosis To be published, Am J Med

36 Bailey, C P Bolton, H E, Jamison, W L Nichols H T, and Likoff, W Methods of Surgical Treatment for Valvular Insufficiency of the Heart, Cardiovascular Surgery, Philadelphia, 1955, W B Saunders Company, p 222

The high frequency of occurrence of thrombosis of the left auricular appendage (29 per cent in 1,200 patients operated on from the left side), and the associated risk of causation of arterial embolization by inadvertent dislodgment of a thrombotic fragment strongly urge the use of some other site of entrance than the appendage into the left atrial lumen for the performance of mitral commissurotomy. The other possible left-sided routes of entry (the pulmonary veins and the atrial wall itself[8, 16]) leave much to be desired both in safety to the patient and in the facility with which the necessary technical procedures may be carried out upon the valve.

The frequency of re-establishment of mitral valvular obstruction, following clinically successful commissurotomy, is related both to the existing pathologic condition of the valve and to the adequacy of the valvular mobilization at the time of the first procedure. The better separation of the valve structures which can be obtained by the right-sided method would seem to commend its initial use in an attempt to obviate the latter etiologic entity. Moreover, once restenosis has become established, a secondary approach to the left atrium through the nearly virginal right thorax and the superior type of valve operation which then becomes feasible would seem to offer the patient the best possible chance for permanent or prolonged relief of the obstruction.

There are many relatively minor advantages associated with the right-sided operation for mitral stenosis which in the aggregate or in the individual case may be of great importance. These include the avoidance of certain left-sided thoracic lesions, the possibility of simultaneously correcting certain right-sided ones, the prevention of severe operative pain and disability which may lead to limitation of shoulder and muscle function, and the superior cosmetic aspects of the right submammary incision.

## CONCLUSION

After an experience comprising the performance of mitral commissurotomy for stenosis from the left side in over 1,400 patients, and from the right side in over 200, it is the opinion of the authors that the latter procedure in most cases is, in highly trained hands, a superior one. This applies both to the initial technical accomplishment upon the valve, and also to the operator's versatility of application with respect to many frequently encountered associated involvements and complications. In comparably severe cases, it appears to be an appreciably safer operation than the left-sided one which requires a left lateral position during surgery. It has been applied repeatedly in individuals who were considered too ill for the older type of operation.

The procedure requires a high degree of technical skill for its proper and safe performance and must not be undertaken by the tyro.

## REFERENCES

1. de Vieussens, R., *In Garrison, F. H.:* An Introduction to the History of Medicine, Philadelphia, 1929, W. B. Saunders Company, p. 250.
2. Laennec, R.: *Traite de l'auscultation mediate,* Paris, 1819 (Translated by John Forbes, 1823).

# INVOLVEMENT OF THE THORACIC WALL IN BRONCHOGENIC CARCINOMA

## STUDY OF 16 CASES IN WHICH PNEUMONECTOMY OR LOBECTOMY AND SIMULTANEOUS RESECTION OF THE THORACIC WALL WERE DONE*

BY K J GRONQVIST, M D,** O THERON CLAGETT, M D,*** AND
JOHN R McDONALD, M D,**** ROCHESTER, MINN

PROGRESS in the treatment of patients who have carcinoma of the lung demands careful appraisal of all the factors affecting treatment and prognosis The literature concerning surgical management of bronchogenic carcinoma with direct extension to the thoracic wall is limited It is hoped, therefore, that this study might add to the understanding of this problem

### REVIEW OF LITERATURE

Coleman, in 1947, reported 5 cases in which pneumonectomy or lobectomy was accompanied by simultaneous resection of the thoracic wall Pneumonectomy was done in 4 of these cases and lobectomy in the remaining one One operative death occurred in his series and the patient who had undergone the lobectomy died 11 months after operation According to Meade 2 of Coleman's patients were still living and well 8 and 13 years respectively, after operation Bjork mentioned 2 cases of this type in his study One of the patients died on the third day after operation and the other survived only 4 months after surgical intervention Overholt and Schmidt, in 1949, stated that their series included 6 cases of this kind, none of their patients survived for more than 27 months Gibbon and associates, in 1953, reported a series of 13 cases in which resection of the thoracic wall was performed, none of these patients survived for more than 18 months

### MATERIAL AND METHODS

The first pneumonectomy for bronchogenic carcinoma at the Mayo Clinic was done in May, 1935 The records of pneumonectomies and lobectomies for bronchogenic carcinoma from that date to November, 1954, were searched in order to find the cases in which simultaneous resection of the thoracic wall was done Sixteen such cases were found, they had been encountered between December, 1947, and October, 1954

---

The Mayo Foundation Rochester Minn is a part of the Graduate School of the University of Minnesota

Received for publication March 22 1956

*Abridgment of thesis submitted by Dr Gronqvist to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Surgery

**Fellow in Surgery Mayo Foundation Roc [illegible] Minn [illegible]

***Section of Surgery Mayo Clinic and Mayo [illegible] for Min [illegible]

****Section of Surgical Pathology Mayo [illegible]

37. Storer, J., Lisan, P., Delmonico, J. E., and Bailey, C. P.: Physiopathological Concepts of Mitral Valvular Disease—Review of 225 Cardiotomies, J. A. M. A. 155: 103, 1954.
38. Bailey, C. P., and Bolton, H. E.: Criteria for and Results of Surgery for Mitral Stenosis, N. Y. State J. of Med., 1956.
39. Jamison, W. L., Rao, K. V. S., and Bailey, C. P.: A Maneuver for Extracting Clots From the Atrium at the Time of Mitral Commissurotomy, With Reports of Four Consecutive Cases, J. THORACIC SURG. 29: 541, 1955.
40. Bailey, C. P., Downing, D. F., Geckler, G. D., Likoff, W., Goldberg, H., Scott, J. C., Janton, O., and Redondo Ramirez, H. P.: Congenital Interatrial Communications: Clinical and Surgical Considerations With a Description of a New Surgical Technique: Atrio-Septo-Pexy, Ann. Int. Med. 37: 888, 1952.
41. Bailey, C. P., Bolton, H. E., Jamison, W. L., and Neptune, W. B.: Atrio-Septo-Pexy for Interatrial Septal Defects, *J. THORACIC SURG. 26: 184, 1953.*
42. Bailey, C. P., Nichols, H. T., Bolton, H. E., Jamison, W. L., and Gomez-Almeida, M.: Surgical Treatment of Forty-Six Interatrial Septal Defects by Atrio-Septo-Pexy, Ann. Surg. 140: 805, 1954.
43. Sondergaard, T., Sorensen, H. R., Poulsen, T., and Anderson, I.: Experimental Production of Atrial Septal Defects Under Direct Vision, Acta. chir. scandinav. 107: 477, 1954.
44. Bougas, J., Musser, B., and Goldberg, H.: Left Heart Catheterization. I. Clinical Methods and Applications, Am. Heart J. 52: 359, 1956.

TABLE I RESECTION OF INVOLVED THORACIC WALL IN BRONCHOGENIC CARCINOMA 16 CASES

| CASE | SEX | AGE (YEARS) | MAIN SYMPTOM AND DURATION | OPERATION* | TYPE OF CARCINOMA | INVOLVEMENT OF INTERCOSTAL NERVES AND VESSELS | FOLLOW UP, TIME POSTOPERATIVE |
|---|---|---|---|---|---|---|---|
| 1 | M | 50 | Pain, 4 mos., | RUL + RTW | Large cell | + | Died 14 mos |
| 2 | F | 63 | Weakness, 4 mos | LLL + RTW | Adenoca grade 4 | Only parietal pleura involved | Living and well, 6½ yrs |
| 3 | M | 47 | Cough, 6 wks | LUL + RTW | Large cell | + | Died 2 mos |
| 4 | F | 66 | Pain, 1 mo | LP + RTW | Adenoca, grade 3 | + | Died 14 mos |
| 5 | M | 63 | Pain 6 mos | RUL + RTW | Squamous cell, grade 4 | + | Died, 1 yr |
| 6 | M | 50 | Pain 2 mos | LP + RTW | Squamous cell, grade 4 | + | Died, 5 mos |
| 7 | M | 65 | Pain 1 mo | RP + RTW | Large cell | + | Died, 3 wks |
| 8 | M | 65 | Pain, 15 mos | LUL + RTW | Squamous cell, grade 4 | + | Died 6 mos |
| 9 | M | 46 | Pain, 2 wks | RP + RTW | Large cell | Only parietal pleura involved | Living and well, 2⅓ yrs |
| 10 | M | 52 | Pain 1 yr | LP + RTW | Mucous adenoca grade 4 | + | Died, 6 mos |
| 11 | F | 82 | Pain, 3 wks | LLL + RTW | Large cell | + | Died 1 yr |
| 12 | M | 68 | Pain 5 mos | RLL + RTW | Squamous cell, grade 4 | + | Living and well 1⅓ yrs |
| 13 | M | 43 | Pain, 5 wks | RUL + sup segm RLL + RTW | Large cell | Only parietal pleura involved | Living and well, 13 mos |
| 14 | M | 35 | Pain, 2½ mos | RUL + RTW | Adenoca, grade 4 | Involvement of brachial plexus | Died 2 mos |
| 15 | M | 60 | Pain, 2 mos | RP + RTW | Squamous cell, grade 4 | + | Living not well 8 mos |
| 16 | M | 66 | Pain 6 wks | RP + RTW | Squamous cell, grade 4 | + | Living and well 5 mos |

*RUL = right upper lobectomy LUL = left upper lobectomy RLL = right lower lobectomy LLL = left lower lobectomy RTW = resection of thoracic wall LP = left pneumonectomy RP = right pneumonectomy

*Bronchoscopic Examination*—Bronchoscopic examination was performed in 7 cases but did not prove to be of help in establishing a definitive diagnosis Some degree of narrowing of the bronchus of the involved lobe was found in 2 cases

*Examination of Sputum*—Malignant cells were found in 4 of the 11 cases in which cytologic studies of the sputum were done

In all cases, gross specimens were examined and sections were made for histologic study. The sections were stained with hematoxylin and eosin. The cases were classified according to the method presented in 1951 by two of us (McDonald and Clagett) and associates.[3, 8, 12, 13] The malignancy of the tumors was graded histologically after the method of Broders. To determine the extent of carcinomatous invasion, multiple blocks were taken in each instance from the parietal pleura and from the intercostal muscle bundles.

In this study, the term "involvement of the thoracic wall" indicates direct and microscopically verified extension of the tumor to the parietal pleura, with or without invasion of the intercostal muscle bundles and ribs.

Frozen sections of the regional lymph nodes are examined routinely at the clinic by the surgical pathologists. These specimens usually are discarded after this examination. Therefore, the presence or absence of carcinomatous involvement of the regional nodes is recorded in this study as noted in the original report of the surgical pathologist.

The clinical features of each case were reviewed and follow-up data were obtained.

## CLINICAL FINDINGS

A summary of some of the data in our 16 patients is presented in the accompanying table (Table I).

*Age and Sex.*—Thirteen of the tumors occurred in men and three in women. The ages of the patients when first seen at the clinic ranged from 35 to 82 years, ut only one of the patients was less than 40 years of age. The average age was 7 years.

*Symptoms and Signs.*—Pain in the chest, back, shoulder, or axilla was the most prominent symptom. It was the main symptom that forced the patient to see the physician in fourteen instances. Pain was preceded by weakness in two instances and by "cold and cough," hemoptysis and osteoarthropathy each in one instance. The average duration of pain before the establishment of the definitive diagnosis was 4 months, with a range of 2 weeks to 15 months. Only 2 of the 16 patients did not complain of pain; one of these had been feeling "all in" for 4 months and the other had had a cough for 6 weeks.

The findings on general physical examination usually were rather indefinite. Some dullness to percussion and diminution in breath sounds on the affected side of the thorax were present in 6 patients. Tender zones on the thoracic wall were found in 2 patients. Results of physical examination of the thorax were essentially normal in the remaining patients, who made up one half of the entire group. No typical examples of Pancoast's syndrome were seen in this series.

*Roentgenographic Findings.*—Rather sharply circumscribed shadows usually were found in roentgenograms of the thorax (Fig. 1, *a* and *b*). Erosion of ribs was detected in 4 cases.

Most of the patients recovered nicely from the operation. Iliofemoral thrombophlebitis developed during the postoperative period in one patient (Case 12) but he recovered. There were no hospital deaths but one patient (Case 7) died 3 weeks after operation.

Several patients complained of an intercostal neuralgia-like pain on the side of the operation for several months after the surgical procedure. It is impossible to estimate the duration of this pain because of the short length of life after the operation in most instances. One patient (Case 2), who is living and well 6½ years after operation, no longer has intercostal pain; another patient (Case 9) who has been followed for 2⅓ years without evidence of recurrence is nearly completely relieved of the pain.

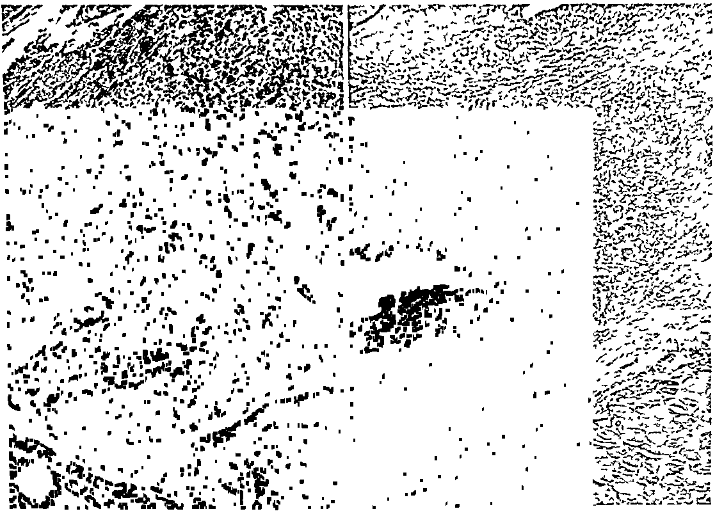


Fig. 2—*a*, (Case 8), Grade 4 squamous-cell carcinoma (hematoxylin and eosin, ×40) *b*, (Case 9), This tumor was a large-cell undifferentiated carcinoma that had involved only the parietal pleura. Note intercostal muscle in right lower corner and parietal pleura in left upper corner (hematoxylin and eosin; ×30).

### RADIATION THERAPY

Three patients (Cases 5, 9, and 13) who did not have any evidence of metastasis received x-ray treatment immediately after their recovery from the surgical procedure. Three other patients (Cases 1, 11, and 14) received radiation therapy for evident metastatic disease. All 3 of these latter patients died of metastasis.

No conclusions can be drawn regarding the efficacy of radiation therapy in the treatment of bronchogenic carcinoma on the basis

## OPERATIVE PROCEDURE AND POSTOPERATIVE MORBIDITY AND MORTALITY

A posterolateral incision was made in each instance on the affected side of the thorax. The sixth rib usually was resected and the pleural cavity was entered through the bed of the resected rib. Pneumonectomy was performed in 7 cases and lobectomy in 9. The involved part of the thoracic wall was resected in continuity with the tumor in 14 instances. En bloc resection was not possible in 2 cases; in one (Case 2), the involved portion of the thoracic wall broke loose from the lung, whereas the tumor in the other (Case 14) had invaded the brachial plexus. In the latter case, the tumor was shaved from the plexus as a palliative procedure.

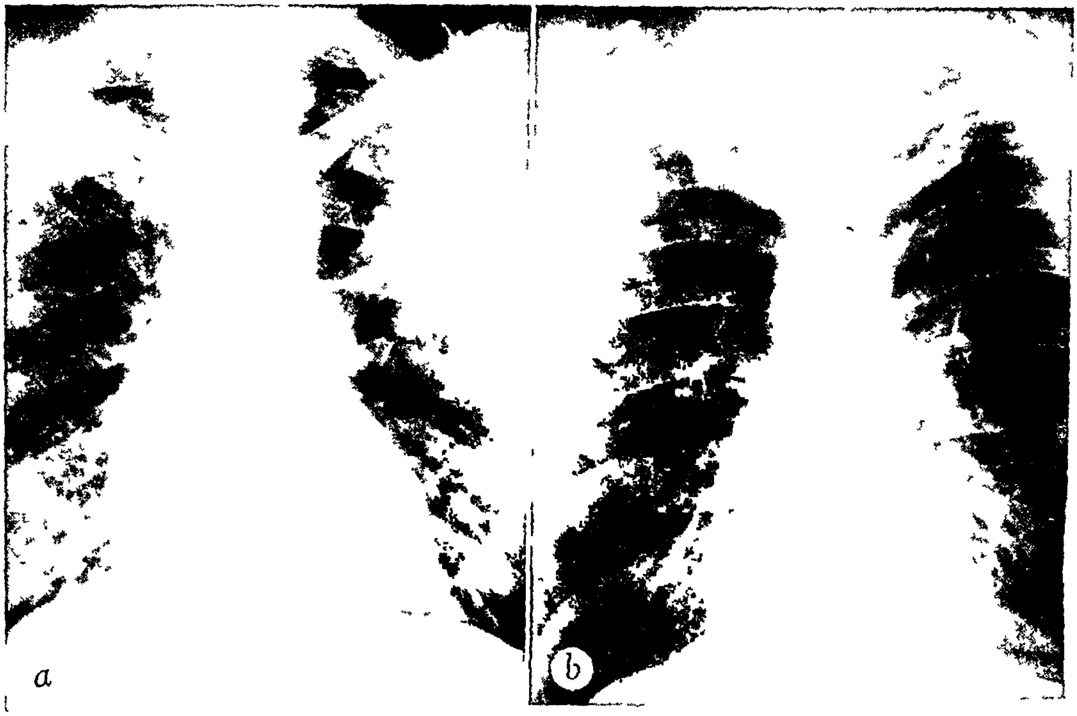


Fig 1.—*a*, (Case 8), Patient had had pain in the left side of the thorax for 15 months Note the large peripheral tumor in the left upper lobe

*b*, (Case 9), This patient had had pain in the right scapula, shoulder, and arm for 2 weeks Note the small tumor in the right apex.

The involved ribs and the intercostal muscle bundles were resected in one piece during resection of the thoracic wall. The resection was carried out at least 2 cm. beyond the edge of the macroscopic invasion by the carcinoma in all but 2 cases, in which such resection was impossible because of additional involvement. The brachial plexus was involved in one of these instances (Case 14), and a vertebra was involved in the other (Case 7). The operation in both of these cases was considered by the surgeon to be palliative in nature

The dead space was filled in 2 instances (Cases 6 and 11) by means of Ivalon sponge. Prolonged postoperative serous drainage developed in one patient (Case 11). This drainage, however, ceased spontaneously. No special measures were used in any other patients to fill the dead spaces and in no instance were any disturbances noted in the healing of the thoracotomy incision.

## LENGTH OF SURVIVAL AFTER OPERATION AND ITS RELATIONSHIP TO EXTENT OF THE LESION

Follow-up data were obtained in all 16 cases. Ten patients have died. The longest survival time in these 10 patients was 14 months (Case 4) and the shortest was 21 days (Case 7). The average length of life after operation in this group was 7 months. The intercostal muscle bundles and the structures in them, including nerves and blood vessels, were invaded by carcinoma in all 10 of these patients. Included were those 4 patients (Cases 1, 5, 8, and 10) in whom erosion of ribs was noted in the preoperative thoracic roentgenograms. The two patients (Cases 7 and 14) in whom carcinoma was known to have been left survived for only 21 and 50 days, respectively.

Four patients (Cases 2, 9, 12, and 13) who have been followed for more than one year were still living at the time of inquiry and had no evidence of recurrence. The survival times in these 4 patients ranged from 13 months to 6½ years (see table). As already noted, 3 of these patients (Cases 2, 9, and 13) had involvement of the parietal pleura only. The intercostal muscle bundles were invaded by carcinoma in Case 12. The regional nodes were not involved in Cases 2, 12, and 13, whereas one hilar node was found to be the site of metastasis in Case 9.

Two other patients (Cases 15 and 16) were living at the time of inquiry but they have been followed less than a year.

## COMMENT

Many authors, such as Jones and co-workers, agree that the 3-year and 5-year survivals after pneumonectomy or lobectomy for bronchogenic carcinoma have been largely among the patients who had peripheral lesions without involvement of the thoracic wall. Jones and associates have been performing lobectomy for peripheral lesions that did not show nodal involvement and were away from the fissures. Twenty (69 per cent) of their 29 patients who underwent lobectomy were living 3 years after operation. On the other hand, their over-all 3-year survival rate was 28 per cent.

Therefore, it is reasonable to believe that peripheral bronchogenic carcinoma with involvement of the thoracic wall may be associated with a favorable prognosis if there has been no spread by the lymphatic system or blood vessels of the thoracic wall. Naturally, definite conclusions cannot be drawn from study of this small series but the available data appear to support this theory. Only 2 patients in our series have survived more than 2 years after operation, neither of these patients presented any evidence of invasion into the intercostal muscle bundles and most likely there was no permeation of the lymphatic system or the blood vessels of the thoracic wall. Symptomatically, this type of an early lesion was associated with malaise of 4 months' duration in one patient and with pain in the thoracic wall of 2 weeks' duration in the other.

Extension into the intercostal muscle bundles and invasion around the intercostal nerves and vessels apparently indicate a poor prognosis. Our patients with such advanced lesions usually had noted pain in the thoracic wall for

PATHOLOGIC FINDINGS

The tumors in this series were all rather sharply circumscribed peripheral carcinomas. Their diameters ranged from 3 to 8 cm.

In 9 of the 16 cases, the tumor was situated in the right lung; the right upper lobe was affected in 7 of these cases and the right lower lobe in 2. Of the seven tumors that occurred on the left side, four were in the left upper lobe and three in the left lower lobe.

Histologically, all the tumors in this series were anaplastic lesions of a high grade of malignancy. Six of them were grade 4 squamous-cell carcinomas (Fig. 2, *a*), six were large-cell undifferentiated carcinomas (Fig. 2, *b*) and the remaining four were adenocarcinomas. One of the adenocarcinomas was a grade 3 lesion and the others were grade 4 carcinomas, one being a mucus-producing adenocarcinoma (Case 10).

The extent of direct invasion of the parietal pleura and the intercostal muscle bundles by the tumors was studied carefully. The carcinomas in 13 cases had invaded extensively into the intercostal muscle bundles surrounding the intercostal vessels and nerves (Fig. 3). As already mentioned, invasion of a vertebra also was present in one instance (Case 7) in addition to the intercostal involvement, and another patient (Case 14) had the additional complication of involvement of the brachial plexus. The intercostal muscle bundles were not involved in three instances (Cases 2, 9, and 13), the invasion including only the parietal pleura (Fig. 2, *b*).

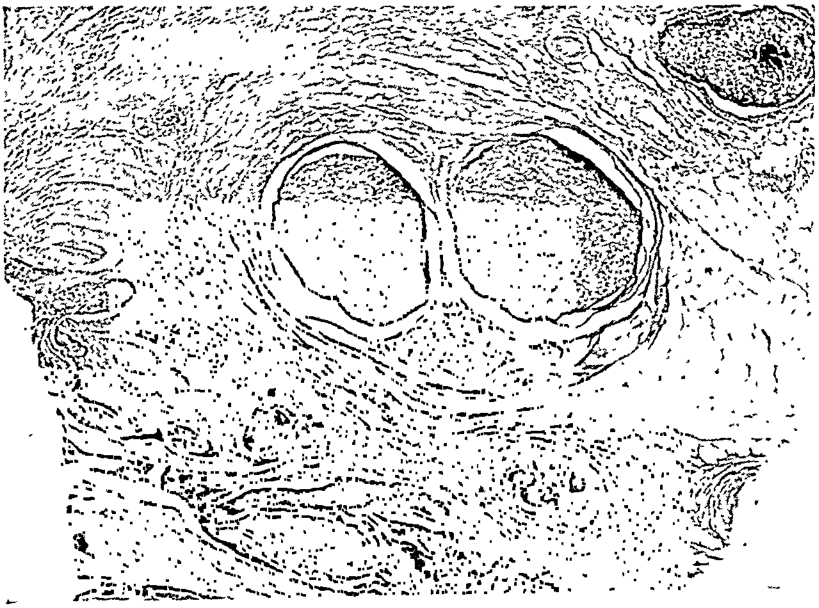


Fig. 3.—(Case 8), Carcinomatous invasion around the intercostal nerves (hematoxylin and eosin; ×30).

The regional lymph nodes were involved in 3 patients (Cases 8, 9, and 16); only one involved node was detected in Cases 8 and 9, whereas massive invasion of the hilar nodes was present in Case 16.

3 Carlisle, J C, McDonald, J R, and Harrington, S W Bronchogenic Squamous Cell Carcinoma, J THORACIC SURG 22 74 82, 1951
4 Churchill, E D Primary Carcinoma of the Lung, J A M A 137 455 461, 1948
5 Coleman, F P Primary Carcinoma of the Lung, With Invasion of the Ribs Pneumonectomy and Simultaneous Block Resection of the Chest Wall, Ann Surg 126 156 168, 1947
6 Gibbon, J H Jr, Allbritton, F F, Jr, Templeton, J Y, III, and Nealon, T F, Jr Cancer of the Lung—An Analysis of 532 Consecutive Cases, Ann Surg 138 498 501, 1953
7 Jones, J C, Robinson, J L, and Meyer, B W Primary Bronchogenic Carcinoma of Lung Statistical Study of Seven Hundred Four Private Patients, Arch Surg 70 265 275, 1955
8 McBurney, R P, McDonald, J R, and Clagett, O T Bronchogenic Small Cell Carcinoma, J THORACIC SURG 22 63 73, 1951
9 Meade, R H Palliative Resection for Cancer of the Lung, In Proceedings of the Second National Cancer Conference, vol 2, part 4, New York, 1952, American Cancer Society, Inc, pp 935 937
10 Ochsner, A, DeBakey, M, and Dixon, J L Primary Cancer of the Lung, J A M A 135 321 327, 1947
11 Overholt, R H, and Schmidt, I C Survival in Primary Carcinoma of Lung, New England J Med 240 491 497, 1949
12 Patton, M M, McDonald, J R, and Moersch, H J Bronchogenic Adenocarcinoma, J THORACIC SURG 22 83 87, 1951
13 Patton, M M, McDonald, J R, and Moersch, H J Bronchogenic Large cell Carcinoma, J THORACIC SURG 22 88 93, 1951
14 Rienhoff, W F, Jr The Present Status of the Surgical Treatment of Primary Carcinoma of the Lung, J A M A 126 1123 1128, 1944

several months before the diagnosis was established. Roentgenographic evidence of erosion of ribs appeared to be an extremely grave prognostic sign. The disease progressed with great rapidity in the 2 patients in whom removal of the entire carcinoma was impossible.

These 16 patients comprise a highly selected group. For this reason, no effort has been made to establish any figures for the incidence of involvement of the thoracic wall or for its operative treatment. It is also unjustifiable on the same grounds to attempt to draw any conclusions as to the dominant type of cell in the lesions invading the thoracic wall. The only significant finding in this regard was the absence of small-cell carcinomas in this group. It has been pointed out previously by McBurney and associates that the small-cell carcinoma is usually a central lesion.

Involvement by carcinoma of the hilar nodes in only 3 of our 16 cases is a noticeably low incidence, in view of the fact that Carlisle and co-workers found nodal involvement in 43.7 per cent of their cases of squamous-cell carcinoma. Ochsner and associates noted nodal metastasis in 60 per cent of their patients in whom resection was done. This incidence was 70 per cent in Rienhoff's series. The low incidence of nodal involvement in the peripheral lesions in our series appears to support the concept that lobectomy may be a satisfactory procedure in carefully selected cases.[4, 7]

## SUMMARY AND CONCLUSIONS

A study has been made at the Mayo Clinic of 16 cases of bronchogenic carcinoma in which involvement of the thoracic wall was present. Pneumonectomy was performed in seven instances and lobectomy in nine. The involved part of the thoracic wall was removed simultaneously in all cases.

The most prominent symptom was pain in the thoracic wall. No cases of Pancoast's syndrome were present in this series.

Six of the sixteen tumors were grade 4 squamous-cell carcinomas, six were large-cell undifferentiated carcinomas and four were high-grade adenocarcinomas. The intercostal muscle bundles and the structures in them were invaded in all but three instances.

Follow-up data were obtained in all cases. Ten patients have died from their disease. Two of the 6 surviving patients have lived more than 2 years (2⅓ years and 6½ years, respectively) after operation. The carcinoma did not extend into the intercostal muscle bundles in either of these 2 patients. The third patient who had involvement of the parietal pleura only was living 13 months after operation.

This study suggests that either pneumonectomy or lobectomy done in association with resection of the thoracic wall for bronchogenic carcinoma can be a curative procedure if the involvement of the thoracic wall is limited to the parietal pleura.

## REFERENCES

1. Björk, V. O.: Bronchiogenic Carcinoma, Acta chir. scandinav. 95 (supp. 123): 1-113, 1947.
2. Broders, A. C.: The Grading of Carcinoma, Minnesota Med. 8: 726-730, 1925.

*Pathology.*—The term, "Pancoast tumor," or "superior pulmonary sulcus tumor," is no longer regarded as a clinical or pathologic entity,[2, 4, 5, 13, 14, 19, 22, 23] and we no longer use it. We still use the term "Pancoast syndrome,"[20] described also by Hare (in 1838), Donald (in 1850), and Tobias[24] (in 1932), to denote the clinical manifestations of a far-advanced chest-wall involvement

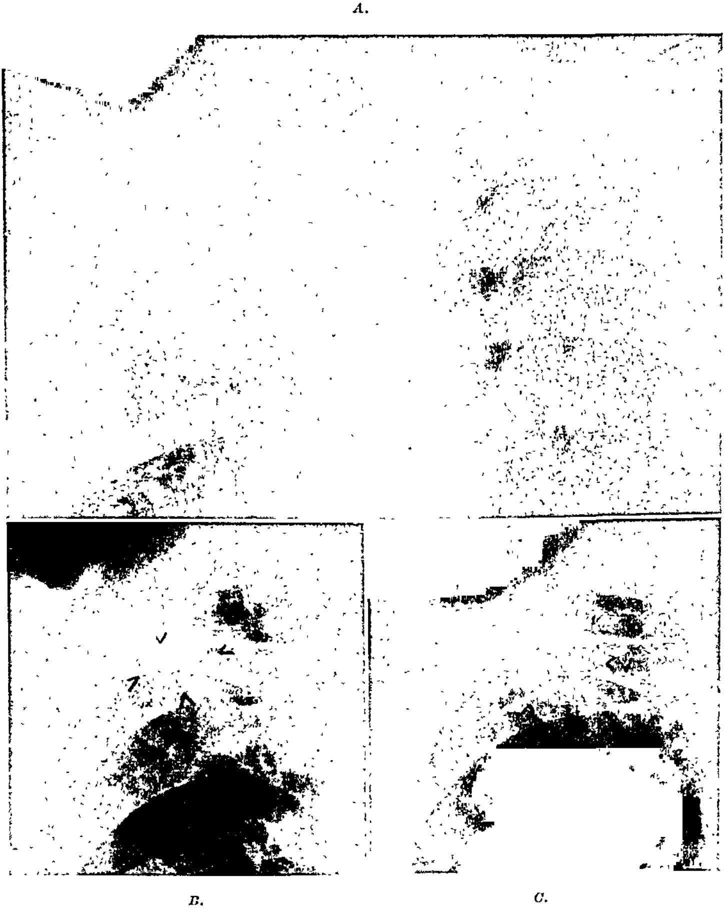


Fig. 1.—Case 11. *A*, At time of diagnosis: extensive lesion of RUL (involving hilar and mediastinal nodes, and second and third ribs).

*B*, Thirty months before diagnosis: hazelnut-sized soft shadow in apex. Primary apical lung lesion (interpreted as recurrence of old tuberculosis).

*C*, Sixteen months before diagnosis: apical shadow increased (still diagnosed as tuberculous lesion).

# RADIATION MANAGEMENT OF APICAL LUNG TUMORS

LEWIS L. HAAS, M.D., ROGER A. HARVEY, M.D., AND CHARLES F. MELCHOR, M.D.
CHICAGO, ILL.

APICAL lung tumors are separated from other lung lesions for this discussion because their clinical and radiotherapeutic behavior is different.

*Terminology.*—Our criteria of apical tumors are:

1. The lesion should arise from the apex.

2. The "apex" is denoted in its topographical meaning and not as the anatomic apical segment. The topographical "apex" implies a peripheral origin and location of the lesion to which the peculiarities of apical lesion are due; the apical segment might also include more central, larger bronchus branches.

3. The lesion should show an early tendency to peripheral invasion, toward the extrapulmonary tissues, because of the relatively small dimension and peripheral locality of the apex.

If apical and subapical areas are involved it may be difficult to differentiate which area was affected primarily. If clinical or radiologic signs of chest-wall involvement (pleural thickening, Horner's syndrome, shoulder-arm pains, rib destruction) developed relatively early, we can assume that the lesion arose from the apex. In late stages, however, nonapical tumors may also extend secondarily into the apical area and produce the same symptoms and signs as apical tumors. Occasionally, the classification may be clarified by available earlier x-ray films. Fig. 1, *A* shows an extensive upper lobe lesion which could be classified only after the patient's chest films of previous years were obtained. These films (Fig. 1, *B* and *C*) clearly localized the initial lesion in the apex. This was interpreted for two and one-half years as recurrent old tuberculosis as the patient had only moderate, vague complaints before the correct diagnosis was made. We classify doubtful cases in the lobar carcinoma group, for example, the case shown in Fig. 2. A massive tumor shadow occupied the apex, with moderate subapical extension. However, dense peribronchial strands leading to the hilum, and the absence of rib destruction and Horner's syndrome aroused suspicion of a central-bronchial origin; bronchoscopy confirmed it by showing the primary lesion close to the bronchial orifice.

4. If subapical areas are also involved, these should be secondary extensions from the apex.

---

From the Department of Radiology, College of Medicine, University of Illinois, Chicago, Ill.
Received for publication March 23, 1956.

We attempted subdivision of the disease according to its extension into the following *stages*.

Stage 1. Pulmonary involvement only.

Stage 2. Involvement of chest-wall soft tissues, including mediastinum, only radiologically or surgically detectable (no Horner's syndrome or bony destruction).

Stage 3. Extension to bones: (a) ribs, and (b) vertebrae.

Stage 4. Paravertebral extension with sympathetic neural involvement, indicated by Horner's syndrome.

Stage 5. Regional or distant metastases.

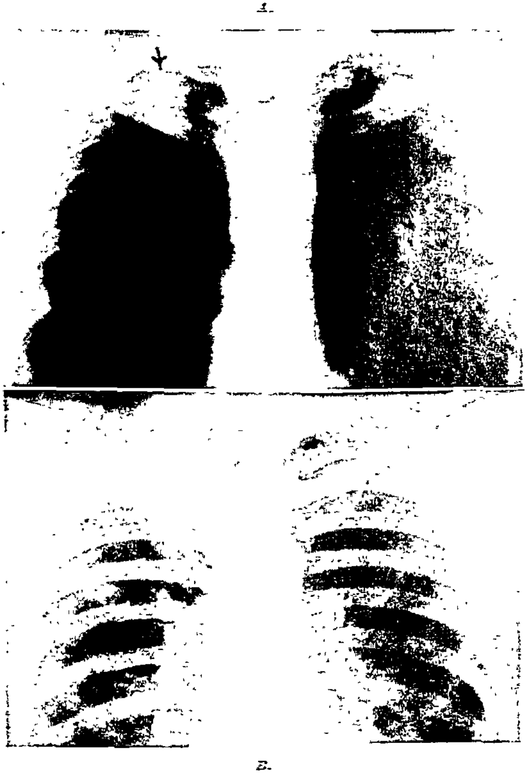


Fig. 2.—Case 3. *A*, Density in right apex, unnoticed in mass survey by rib superimposition.

*B*, Thirty months later: intrapulmonary mass with pleural involvement; complete segmental resection.

in the apical region which invades the sympathetic ganglion chain in the paravertebral area. Thereby, the lesion may arise initially from any part of the apical or subapical areas of the lung or of the extrapulmonary tissues, and its histopathology may be very diversified.[2, 18, 19, 22]

In the literature, a few authentic cases[1, 3, 7, 10] of primary epithelial tumors of the thoracic inlet without lung involvement are recorded. These tumors arose from unknown extrapulmonary tissues, but the great majority of those described originate from terminal lung bronchioles.[1, 13, 15, 23] As far as we could determine in our cases by radiologic or surgical means, or on surgical pathologic specimens, the lung was always involved with pleural or peripulmonary extensions at the time of diagnosis. It is in general impracticable to determine whether the pulmonary or the extrapulmonary lesion is primary. However, the x-ray films, the histopathology, where available, and the clinical course favored the assumption that all tumors in our cases arose from the apical lung parenchyma. One can assume an early stage not yet involving the chest wall (see Fig. 1), as well as an intermediate stage with chest-wall involvement but without bony and neural invasion. This is illustrated by Fig. 3. The available minifilm of June, 1949, was negative, but that of June, 1952 (Fig. 3, *A*), shows a density in the center of the apex, presumably in the lung, which was unnoticed because of rib superimposition. Diagnosis was made only two and one-half years later, in November, 1954, also by a mass survey film (Fig. 3, *B*). The patient still had no clinical signs or symptoms, except for occasional vague shoulder discomfort, which was revealed only by direct questioning. Roentgenograms and surgical resection showed a pulmonary mass with involvement of posterior and mediastinal pulmonary walls which could not be entirely removed. The cases illustrated by Figs. 1 and 3 indicate how long the tumor may be present before the diagnosis is made.

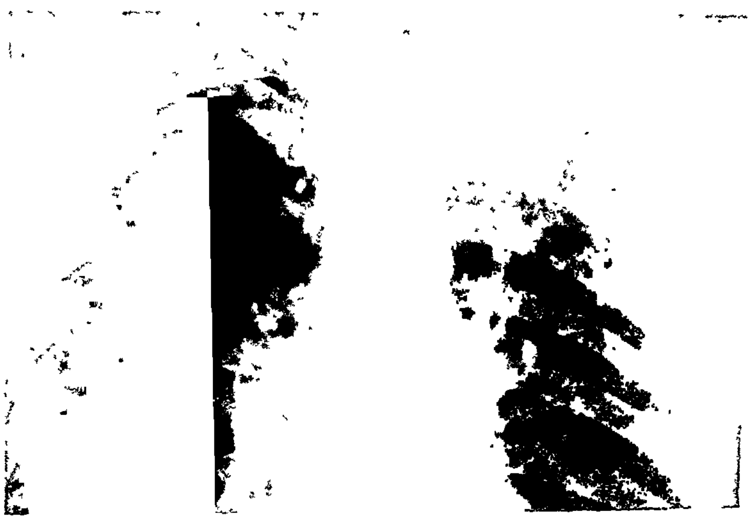


Fig. 2.—Tumor mass in left apical and subapical area; dense peribronchial strands leads to the hilum. Classified as lobar, not as apical, peripheral lung carcinoma. Primary lesion was found close to the upper bronchus orifice.

The reports of 8 cases, treated by *conventional x rays* over the past twelve years, were found in our files and details are given in Table I. We treated 10 patients in six years by 22.5 million volt *betatron x rays*, the details of which are outlined in Table II.

TABLE II DATA OF CASES TREATED BY 22.5 MEV BETATRON X RAYS

| F<br>E | SMOKING | HISTOLOGY | SYMPTOMS | WEIGHT LOSS (LBS.) | SIDE | STAGE | INVOLVEMENT | | | | DURATION (MONTH) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | PLEURAL CHEST WALL | BONY | NEURAL | METASTASES | FIRST SYMPTOM TO FIRST DIAGNOSIS | FIRST DIAGNOSIS TO FIRST IRRADIATION |
| G<br>5 | ++ | Epidermoid carcinoma with adenocarcinoma qualities | Slight arm pain, slight cough | 12 | R | 2 | + | 0 | 0 | 0 | 30 (mini film) | 3 |
| B<br>3 | + | Aspiration + | Chest pain, disabled, weak | 20 | R | 3a | + | + | + | 0 | 10 | 2 |
| C<br>7 | ++ | Class IV cells | Chest pain, cough, hemoptysis, weak, disabled | 20 | R | 3a | + | + | 0 | 0 | 32 (Tbc diagnosis) | 1 |
| R<br>3 | ++ | ? | Back pain, cough, weakness, disabled | 25 | R | 3b | + | + | ± | 0 | 21 | 3 |
| M<br>0 | ++ | Epidermoid carcinoma | Arm pains, numb fingers, neck fullness | ? | L | 4 | + | + | + | 0 | 6 | 5½ |
| H<br>7 | + | ? | Shoulder pain, neck swelling, vena cava syndrome, disabled | 12 | R | 4 | + | + | + | 0 | 5 (Osteop treatment) | 3 wks |
| J<br>8 | ? | Adenocarcinoma | Shoulder arm pain | 23 | R | 5 | + | 0 | 0 | + brain hilus | 6 (Chir opr treatment) | 2 |
| C<br>0 | ++ | Poorly differentiated carcinoma | Chest pain, cough dyspnea | 30 | R | 5 | + | + | ? | + skin | 12 (Tbc diagnosis) | 1 |
| A<br>1 | ? | Adenocarcinoma | Shoulder arm pains, anorexia, weak, disabled, neck nodes | 9 | L | 5 | + | + | + | + neck | 8 | 1 |
| E<br>5 | 0 | Epidermoid carcinoma | Shoulder arm pain, hemoptysis, neck swelling, weak, disabled | ? | L | 5 | + | + | ? | + skin | 2 | 3 |

It is not essential that all symptoms of the Pancoast syndrome be present in early stages.[9, 19, 23] The most constant and earliest indication is pain, occurring early after involvement of the parietal pleura, or intercostal, or brachial plexus nerve branches. The Horner syndrome may appear later after encroachment of the sympathetic chain, but it may remain absent if the same lesions extends laterally and avoids the paravertebral area. Wasting of the muscles of the hand, secondary to disuse or to neural involvement and emphasized by Pancoast, was usually a late finding in our cases. Rib destruction was also absent initially in some cases. However, the absent signs may develop later, after the diagnosis has been made, following the extension of the tumor.

## MATERIAL

Our apical tumor patients were irradiated, in part, (a) by conventional x-rays and, in part, (b) by high energy x-rays of the betatron.

Table I. Data of Cases Treated by Conventional X-rays

| | | | | | | | INVOLVEMENT | | | | DURATION (MONTHS) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | SMOKING | HISTOLOGY | SYMPTOMS | WEIGHT LOSS | SIDE | STAGE | PLEURAL CHEST WALL | BONY | NEURAL | METASTASES | FIRST SYMPTOM TO FIRST DIAGNOSIS | FIRST DIAGNOSIS TO FIRST TREATMENT |
| | ? | Carcinoma, minimal evidence (aspiration) | Chest pain, cough | + + | L | 2 | + | 0 | 0 | 0 | 4 | 3 |
| | + + | Class IV cells (aspiration) | Chest pain, arm pain, cough, dyspnea | + + | R | 3a | + | + | 0 | 0 | 4 | 3 |
| 66 | + + | Poorly differentiated carcinoma | Chest pain, cough, hemoptysis | ? | R | 3b | + | + | 0 | 0 | 17 | 2 |
| 4.<br>S. A.<br>49 | ? | Carcinoma | Shoulder pain | ? | L | 4 | + | 0 | + | 0 | ? | 1 |
| 5.<br>K. C.<br>63 | + | ? | Shoulder pain; chest pain, cough, dyspnea | ? | R | 4 | + | + | + | 0 | 4 | 1 |
| 6.<br>C. W.<br>62 | + + | Poorly differentiated carcinoma | Chest pain, cough, neck mass | ? | R | 5 | ? | 0 | 0 | + neck | 10 days | 2 |
| 7.<br>M. J.<br>46 | + | Undifferentiated carcinoma | Shoulder pain, chest pain, leg numbness, hoarseness, neck mass | + + | L | 5 | + | 0 | + | + neck | 5 | 10 day |
| 8.<br>M. R.<br>54 | ? | Carcinoma simplex | Shoulder pain, neck nodes | ? | L | 5 | + | + | + | + neck | 12 | 1 |

Table VI shows the anatomic involvement at the time irradiation was started

TABLE VI ANATOMIC EXTENT OF TUMOR AT BEGINNING OF RADIATION

| | CONVENTIONAL X-RAY THERAPY | BETATRON THERAPY | TOTAL |
|---|---|---|---|
| *Side* | | | |
| Right apex | 4 | 7 | 11 |
| Left apex | 4 | 3 | 7 |
| *Stage* | | | |
| 1 | - | - | - |
| 2 | 1 | 1 | 2 |
| 3a | 1 | 2 | 3} 5 |
| 3b | 1 | 1 | 2} |
| 4 | 2 | 2 | 4 |
| 5 | 3 | 4 | 7 |
| *Extension* | | | |
| Pulmonary cavitation | 2 | 1 | 3 |
| Bones Ribs | 4 | 8 | 12 |
| Vertebrae | 2 | 5 | 7 |
| Sympathetic chain | 4 | 3 + 2 | 7 + 2 |
| Supraclavicular protrusion | 1 | 2 | 3 |
| Engorged veins | - | 1 | 1 |
| Neck node | 3 | 2 | 5 |
| Hilar or mediastinal node | 1 | 3 | 4 |
| Distant metastasis | - | skin 2}<br>brain 1} | 3 |

The right side was more frequently involved than the left side (11 7)

Using our staging (p 499), no patient in Stage 1 was treated (without extrapulmonary involvement) because the diagnosis was not made earlier In only 2 cases did reinspection of prediagnostic x rays suggest the occurrence of apical lesion without extrapulmonary extension Two patients were treated who did not have bony or sympathetic nerve or metastatic involvement, but who had surgically verified chest wall invasion (Stage 2) In 5 cases, bony destruction was present without signs of sympathetic nerve or metastatic involvement (Stage 3) In 4 cases Horner's syndrome was present without metastasis (Stage 4), and in 7 cases metastases were already found either in the regional nodes or in distant areas (Stage 5)

According to anatomic structures, pleural involvement was assumed in 16 cases and could not be diagnosed in 2 cases, although both patients already had metastases Destroyed ribs were found in 12 cases vertebral erosion in 7 cases The rib involvement started frequently as a pressure erosion, marked radiologically by notching with maintenance, later with loss of cortical layers (Figs 4 and 5) Rib lesions may be demonstrated frequently better and earlier on oblique films (Fig 5), particularly in the paravertebral area Actual invasion of the bone by tumor was probably a secondary process (Fig 6, *A C*), but the two stages could not be classified separately Sympathetic chain involvement was diagnosed in 9 cases (in 7, complete Horner's syndrome, in 2, only anisocoria) Supraclavicular protrusion of the apical tumor was seen in 3 cases in one case with engorgement of the neck and chest wall veins (Fig 7, *A D*) Metastatic neck nodes were found in 5 patients, mediastinal or hilar nodes in 4, and distant metastasis in 3 (2 skin, 1 brain)

The 18 cases are further summarized as follows: Table III shows that all patients were white males, except one white female (Case 18). Of 13 patients with recorded smoking habits, 8 were heavy smokers (over one package of cigarettes daily), 4 moderate smokers (one package or less per day); the one female did not smoke.

TABLE III. DISTRIBUTION BY AGE, SEX, RACE, SMOKING HABITS

| | CONVENTIONAL X-RAY THERAPY | BETATRON THERAPY | TOTAL |
|---|---|---|---|
| Age | 46-66 yrs.<br>(average 56.6) | 51-67 yrs.<br>(average 59.9) | 46-67 yrs.<br>(average 58.4) |
| Sex | | | |
| Male | 8 | 9 | 17 |
| Female | - | 1 | 1 |
| Race | | | |
| White | 8 | 10 | 18 |
| Negro | - | - | - |
| Smoking habits | | | |
| Heavy | 3 | 5 | 8 |
| Moderate | 2 | 2 | 4 |
| Nonsmoker | - | 1 | 1 |
| Unknown | 3 | 2 | 5 |

Table IV shows that the histopathologic distribution of the 10 cases determined is similar to that of other bronchogenic cancers.

TABLE IV. HISTOPATHOLOGY

| | CONVENTIONAL X-RAY THERAPY | BETATRON THERAPY | TOTAL |
|---|---|---|---|
| Epidermoid carcinoma | - | 2 | 2 |
| Epidermoid carcinoma with adenocarcinoma qualities | - | 1 | 1 |
| Adenocarcinoma | - | 2 | 2 |
| Undifferentiated—poorly differentiated carcinoma | 3 | 1 | 4 |
| Simplex carcinoma | 1 | - | 1 |
| Undetermined type or not examined | 4 | 4 | 8 |

Table V shows the presenting symptoms at the time of irradiation. Shoulder, arm, and chest pains were the most frequent complaints, but weight loss, weakness, and cough also occurred frequently. Atrophy of the muscles of the hand was usually either absent, or mild when first seen, and thus not recorded in several histories.

TABLE V. SYMPTOMS AT BEGINNING OF RADIATION

| SYMPTOM | CONVENTIONAL X-RAY THERAPY | BETATRON THERAPY | TOTAL |
|---|---|---|---|
| Shoulder-arm pain | 5 | 6 | 11 |
| Chest pain | 5 | 4 | 9 |
| Numbness in fingers or toes | 1 | 1 | 2 |
| Weight loss | 3 | 8 | 11 |
| Weakness, disability | 2 | 6 | 8 |
| Cough | 4 | 4 | 8 |
| Dyspnea | 2 | 1 | 3 |
| Hemoptysis | 1 | 2 | 3 |
| Hoarseness | 1 | - | 1 |

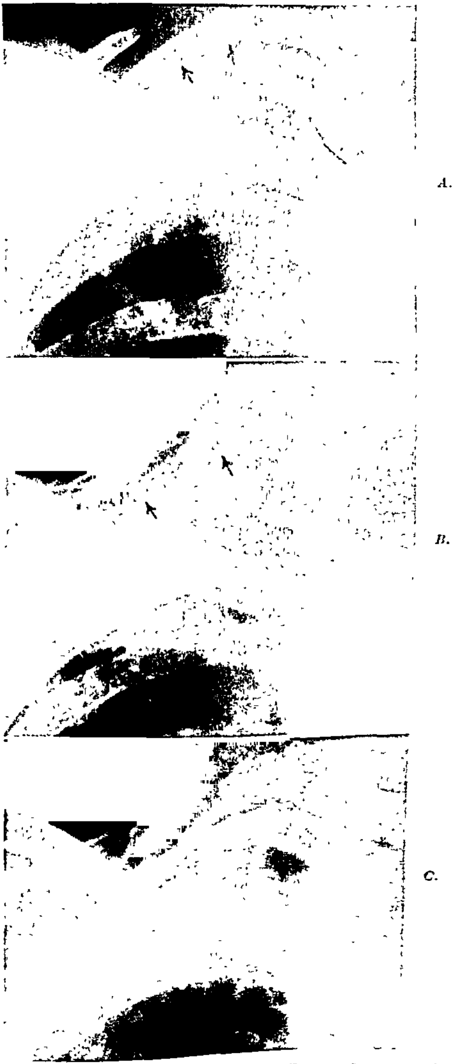


Fig. 6.—Case [illegible] of the second rib with largely maintained cortical outline. Begin[illegible] *B*, Three weeks later, cortical layer still present medially. In [illegible] of the erosion actual bone invasion occurred with destruction of [illegible] sixteen months after begin[illegible] rib, radiation [illegible]

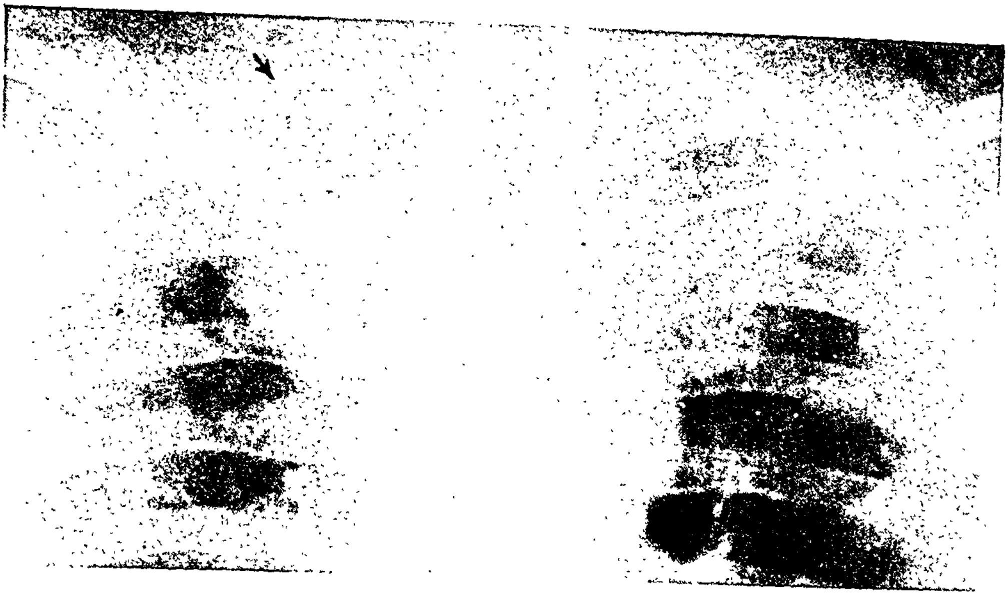


Fig. 4.—Case 5. Notching of the first rib, with visible cortical layer.

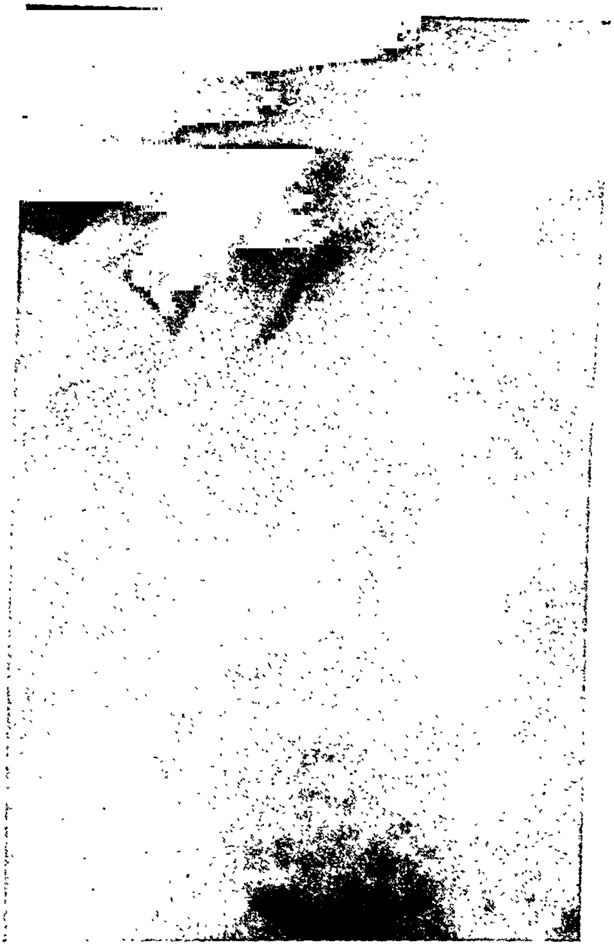
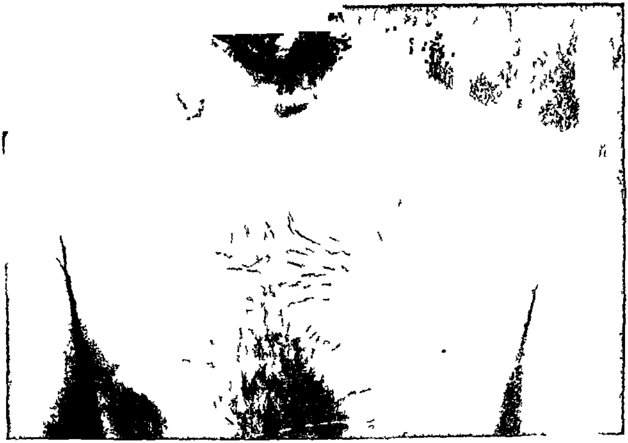


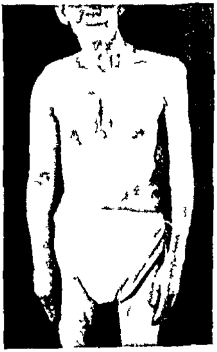
Fig. 5.—Case 16. Local indentation of first rib with loss of cortical layer. Diffuse erosion of the posterior lower margin of the second rib, with partially maintained cortical layer.

TABLE VII. DURATION OF SYMPTOMS OR SIGNS BEFORE DIAGNOSIS AND BEFORE FIRST IRRADIATION

| | CONVENTIONAL X-RAY THERAPY | | ROTATION THERAPY | | TOTAL | |
|---|---|---|---|---|---|---|
| | MONTHS | AVERAGE (MONTHS) | MONTHS | AVERAGE (MONTHS) | MONTHS | AVERAGE (MONTHS) |
| From first symptom to first diagnosis | ½–17 | 7.0 | ½–32 | 12.3 | ½–32 | 10.1 |
| From diagnosis to first irradiation | ¼–3 | 1.7 | 1–5½ | 2.2 | ¼–5½ | 2.0 |



*C*



*D*

Fig. 7.—Cont'd. *C*, After 1¼ years supraclavicular fossa is filled up by firm fixed fibrotic tissue (radiation fibrosis); in the center biopsy scar. Residual venous dilatations visualized by infrared photography.

*D*, After 3 years retraction of right upper thorax by pulmonary fibrosis, muscular atrophy of the shoulder arm and paresis of the hand due to damage of brachial plexus by cervical radiation fibrosis.

Table VII shows the duration of symptoms or signs prior to diagnosis, and the interval from diagnosis to the first irradiation. These data show that the former interval in several cases was as long as one year, up to 32 months, in average over 10 months; that the latter interval was as much as 5½ months, in average 2 months. The prediagnostic delay was due in part to faulty diagnosis and therapy, preradiation delay referable mainly to intervening surgical procedures (resection, exploration). Thus, the irradiation was started in average 12 months after onset.

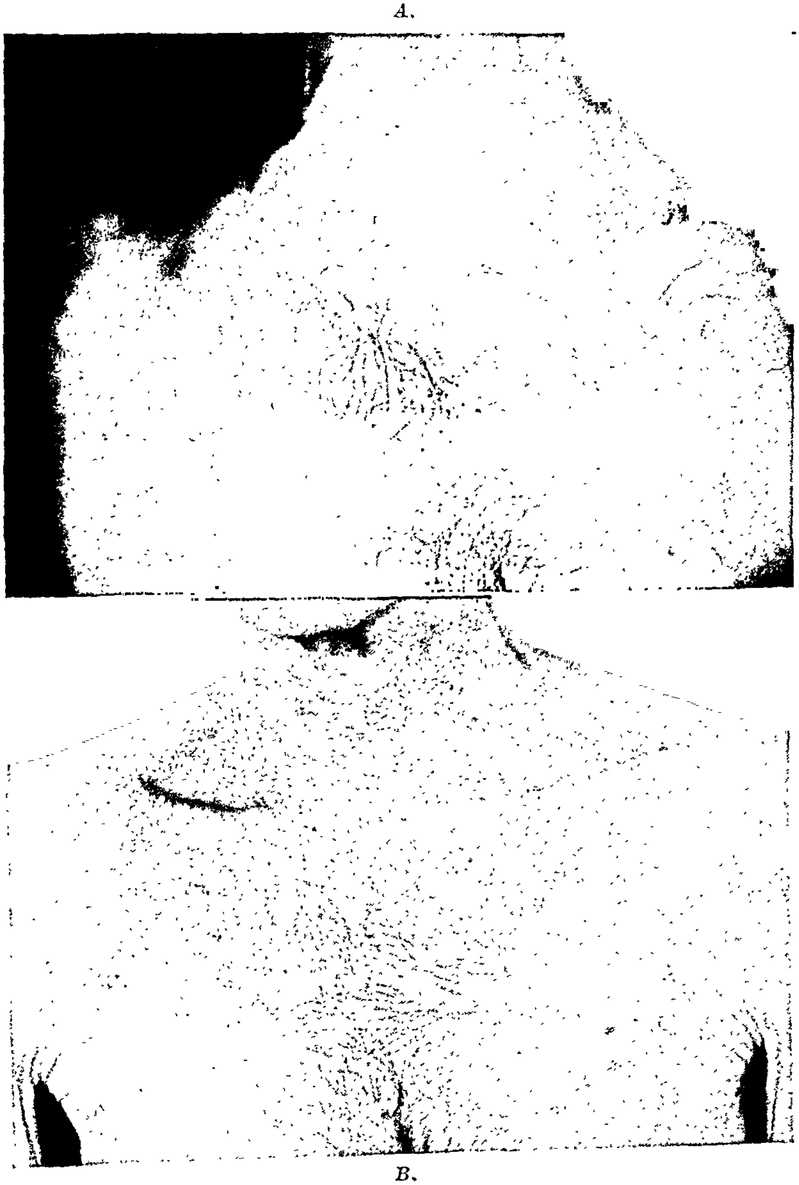


Fig. 7.—Case 14. *A*, Supraclavicular protrusion of apical tumor, engorgement of neck and chest-wall veins. *B*, After completing betatron irradiation: regression of protrusion and of engorgement. Deepened supraclavicular fossa. Moderate pigmentation.

## RADIOTHERAPY

The technique and results of irradiation are considered separately for groups treated with conventional x-rays and with betatron x-rays.

is living and well after 11 years. The aspiration biopsy was reported as "carcinoma, diagnosis based on minimal evidence." Histologic slides and preoperative x-rays are not available for review so we accept the diagnosis with caution. However, the characteristic description of the pulmonary and inoperable extrapulmonary invasion from the exploratory surgical findings leave little doubt of the diagnosis. We regard this case as one of the rare, but known curiosities with exceptional healing tendency. The other 7 cases had survivals after the first irradiation of 3 to 9 months with an average of 6 months. The average survival according to the stages was 7.5 months for Stages 3 and 4, and 4 months for Stage 5.

The survival time after the first symptoms in 6 of the cases was 5 to 26 months, averaging 14 months, and after diagnosis 5 to 12 months with an average of 7.4 months.

TABLE VIII. DURATION OF LIFE

| | | | | | SURVIVAL AFTER | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | ONSET | | DIAGNOSIS | | RADIATION | |
| STAGE | GROUP | NUMBER OF CASES | LIVING AND WELL | DECEASED | MONTHS | AVERAGE | MONTHS | AVERAGE | MONTHS | AVERAGE |
| 2-4 | Conventional x-ray treatment | 5 | 1 | 4 | 11-26 | 17.7 | 7-12 | 9.3 | 6-9 | 7.5 |
| 2-4 | Betatron x-ray treatment | 6 | 2 | 4 | 29-54 | 43.6 | 19-49 | 33.1 | 17-48 | 32.4 |
| 5 | Conventional x-ray treatment | 3 | - | 3 | 5-17 | 10.7 | 4-5.5 | 4.8 | 3-5 | 4.0 |
| 5 | Betatron x-ray treatment | 4 | - | 4 | 8-20 | 15.7 | 6-10 | 8.7 | 3-8 | 6.7 |
| Total | Conventional x-ray treatment | 8 | 1 (11 yrs) | 7 | 5-26 | 14.0 | 5-12 | 7.4 | 3-9 | 6.0 |
| Total | Betatron x-ray treatment | 10 | 2 (11, 27 months after treatment) | 8 | 8-54 | 29.7 | 6-49 | 21.0 | 3-48 | 19.6 |
| Literature | | | | | | 12.0 (nonirradiated, 9.7, irradiated, 14) | | 6.0 | | 6.0 |

*Technique.*—

1. *Conventional x-rays:* In the 12-year period, different techniques were used by different therapeutists in the 8 cases. One incompletely reviewed case is apparently cured for over 11 years (see later). This case was irradiated with 400 kv. 2.75 mm. copper half value layer, in 3 fields of 10 by 10 cm., 50 cm. target skin distance, 9,150 r total air dose in 60 days. A rough estimation of the dose to the tumor center was 5,300 r.

Among the other 7 cases, 200 kv., 0.9 mm. copper half value layer was used in one case, 215 kv., 1.05 mm. copper half value layer in one; 400 kv., 2.75 mm. copper half value layer in 3 cases, 400 kv. with grid in 2 cases. The calculated or retrospectively estimated tumor dose varied between 4,200 and 7,500 r, the over-all treatment time between 30 and 129 days.

2. *Betatron therapy:* Ten cases were treated with 22,500,000 volt x-rays of the betatron with multiportal technique, 4 to 8 ports being employed. After adequate experiences and systematic isodose curve analysis, we reduced the number of fields, usually to 5. The field size varied, according to the shape and dimensions of the patient and to the extent of the lesion, from an 8 to 10 cm. square portal to a 15 cm. circle. The over-all treatment time varied between 26 and 44 days. The shorter times were for technical reasons in our early experimental period but the majority were within 40 to 43 days. The total dose given to the center of the tumor was 8,000 to 11,000 betatron r; the dose in the tumor as found by isodose curves varied between +4 and −8 per cent. We included the paravertebral area and the lateral side of the vertebrae in the fields as much as possible and we tried to avoid the spinal cord. However, we now feel that a greater part of the vertebrae should be included, if paravertebral invasion can be assumed, because some residuals and recurrences were found at autopsies in the prevertebral and cord areas in spite of healed pulmonary and peripulmonary lesions. Moreover, the radiologic examinations in some other cases showed progression of the vertebral lesion, with regression of the pulmonary lesions. We observed spinal symptoms in 2 cases after radiation. In 4 cases, following completion of a full radiation course with inadequate response, or with recurring symptoms, and after some hesitation, we gave a second series of treatments, taking into account the risk of radiation damage. Our philosophy was that it would be preferable for the patient to die from possible late radiation damage after an additional 1 to 2 years of useful life, than in a few short months from uncontrolled disease. One of these patients had a useful survival for 18 months after the second series, and died in 10 weeks of spinal symptoms (see Fig. 11). One other patient survived 26 months after the second series, with 24 months of useful life, dying of spinal and cerebral metastases (see Fig. 12). Both had a good response for 12 and 4 months, respectively, after the first radiation series. In the other 2 cases, in which response to the first radiation course was slight, or of short duration, the second series was also a failure.

## RESULTS

*A. Survival.*—

1. *Conventional x-ray treatment:* As mentioned before, one case (Stage 2)

B *Palliation* —

The criteria of results include not only curability or prolonged survival time but also palliative effects. Important in regard to palliation are the ways in which radiation helps the patient live with his disease during his remaining life. Significant accomplishments of this type consist of a decrease of suffering, a postponement and shortening of the terminal stage, ability to return to a useful family, social, and professional life for any length of time thereby getting the benefit of doubt, and acquisition of a hopeful psychologic attitude to life during a limited life span in cases where prolonged life can not be expected to result.

1 *Conventional x ray cases.* Of the 7 deceased patients 5 were failures showing no significant change in progression of the disease. Two cases improved temporarily. In one case, moderate regression of the complaints and of neck nodes was observed, but the patient died of complications of cardiac and prostatic disease. In the other case with gross vertebral erosions the palliation was marked, all symptoms significantly improved for about 7 months, the pulmonary cavitation which also penetrated the chest wall decreased and destroyed ribs partially regenerated (Fig. s 1 *D*) but the patient died 9 months after the first treatment following a rapid preterminal downhill course of 4 weeks.

2 *Betatron treated cases.* Of the 10 cases, 2 are alive in practically asymptomatic conditions. Of the 8 deceased patients, the palliation in one was insignificant, whereas in all other 7 cases it was very marked. These patients became asymptomatic or showed very marked improvement so that they could return to a useful life of 16 to 44 months in Stages 3 and 4 but only for 1 to 3 months in Stage 5. In Stage 5 life was not significantly prolonged but the relief of suffering, even though temporary, was so striking that we would not deny this assistance to the patients.

The first and most frequently observed effect was the relief of incapacitating *pains in shoulder*, arm, and hand. This effect was observed at times as early as the second day of irradiation, leading us at first to believe in some cases, for instance, in Case 10 that it was a psychologic one, until its objectivity was proved by continuity of gradual pain relief. This pain practically subsided in 5 of our 6 cases, but in one case the relief was only temporary and moderate.

The *chest pain* subsided somewhat later, usually parallel to the radiologic regression of the lesions and in all 5 cases where it was a presenting symptom.

The relief of pain was probably in great part the reason that 5 of our 6 weakened disabled patients, some brought in by wheel chair, shortly regained their strength, became self dependent, could again use their arms and could resume their normal life completely or with some limitations for variable intervals according to their further course.

*Cough, hemoptysis, dyspnea* also subsided, if present, parallel with the tumor regression.

*Weight gain* was a rather objective indicator of tumor regression. At times the original weight was exceeded. Recurring weight loss was usually a premonitory sign of recurrence, metastasis, or postradiative t

2. *Betatron therapy:* Of the 10 patients treated with irradiation, 2 are living and well over 11 and 27 months, respectively, since the first irradiation, 43 and 60 months since the first symptoms.

The 8 deceased patients survived 3 to 48 months, with an average survival of 19.6 months after the first irradiation, 29.7 since the first symptoms. As to the staging, the survival after the first irradiation was 17 to 48 months, with an average of 32.4 months in Stages 3 and 4; and 3 to 8 months, averaging 6.7 months, in Stage 5 with regional or distant metastasis. This big difference in survival corroborates the old experience that if one lymphatic area is invaded, seeding is usually already present in other lymphatic pathways as well.

As noted, the survival time in Stages 2 to 4 was much longer in the betatron treated cases, averaging 32.4 months, against 7.5 months in the conventional x-ray cases. In Stage 5, this difference was only moderate, 6.7 months versus 4 months. It seems that the survival time does not vary appreciably for Stage 5 patients regardless of whether treated or untreated, or how irradiated.

These data of survival are summarized in Table VIII.

The therapeutic data of the treated patients concerning dosage, radiation effects, and final outcome are comprised in Tables IX and X.

TABLE IX. THERAPEUTIC DATA OF CASES TREATED BY CONVENTIONAL X-RAYS

| CASE | RADIATION C. TUMOR DOSE R IN DAYS | SURVIVAL AFTER | | EFFECT | | REMARKS |
|---|---|---|---|---|---|---|
| | | ONSET (MONTHS) | FIRST RADIATION (MONTHS) | CLINICAL | X-RAY | |
| 1 | 5300 r<br>65 days | 11 yrs. | 11 yrs. | Cured | Cured | Explored: inoperable chest wall and mediastinal invasion, living and well over 11 yrs. |
| 2 | 7500 r<br>45 days | 16 | 9 | All symptoms moderately improved | Decreased tumor, cavity, rib regeneration | Cavitation in tumor, perforated to chest wall |
| 3 | 4700 grid<br>129 days | 26 | 7 | 0 | 0 | Cavity in tumor |
| 4 | 4200 r<br>39 days | ? | 8 | 0 | Some tumor decrease | Incomplete segmental resection, died of frontal lobotomy |
| 5 | 4200 r<br>33 days | 11 | 6 | 0 | 0 | |
| 6 | 4400 r<br>grid<br>30 days | 5 | 3 | Slight improvement, decreased neck mass, subjectively improved | ? | Died of heart and prostatic complications |
| 7 | 6500 r<br>127 days | 10 | 5 | 0 | ? | Also hilar nodes irradiated |
| 8 | 5800 r<br>59 days | 7 | 4 | 0 | 0 | Died of spinal and brain metastases |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| 14 | 11,000 r<br>26 days | 54 | 48 | Asymptomatic, temporary | Tumor fibrosed, rib regeneration | 14 | 2 weeks | Posttreatment neck induration, disturbed arm function, died after alcoholic avitaminosis with rapid cachexia and dysphagia, no tumor evidence |
| 15 | 9 100 r<br>38 days | 15 | 7 | Pains gone, weight gain, diplopia | Tumor fibrosed, skull metastases | 3 | 3 | Autopsy no tumor in thorax, cerebral metastases |
| 16 | I 8 030 r<br>40 days<br>II 4,000 r<br>24 days | 22 | 9 | I Almost asymptomatic<br>II 0 | I Cavity gone, tumor fibrosed | 2 | ? | Cavitation of tumor, died of vertebral spinal involvement |
| 17 | I 9 650 r<br>39 days<br>II 6 500 r<br>19 days | 18 | 8 | I Slight decrease pains<br>II 0 | 0 | 4 weeks | 3 | Autopsy fibrotic apex with small residual tumor nodules, died of generalized metastases |
| 18 | 10,000 r<br>43 days | 8 | 3 | Almost asymptomatic | Lung tumor regressed, bony lesions progressed | 2 | 1 | Exploratory operation, died of generalized metastases |

TABLE X. THERAPEUTIC DATA OF CASES TREATED BY 22.5 MEV BETATRON X-RAYS

| CASE | RADIATION | SURVIVAL AFTER |  | EFFECT |  | USEFUL LIFE (MONTHS) | TERMINAL STAGE (MONTHS) | REMARKS |
|---|---|---|---|---|---|---|---|---|
|  | C. TUMOR DOSE R IN DAYS | ONSET (MONTHS) | FIRST RADIATION (MONTHS) | CLINICAL | X-RAY |  |  |  |
| 9 | 8,400 r<br>42 days | 43 | 11 | Asymptomatic, 20 lbs. gain | -- | 11<br>Living and well | -- | Diagnosis by mass survey, incomplete *resection* |
| 10 | 9,100 r<br>31 days | 29 | 17 | Asymptomatic 15 lbs. gain | Tumor fibrosis, rib regeneration | 16 | 16 days | Died of heart symptoms, cardiac involvement? |
| 11 | 8,000 r<br>44 days | 60 | 27 | Asymptomatic | Lung fibrosis, rib regeneration | 27<br>Living and well | -- | Extensive UL lesion to hilum.<br>Posttreatment: pulmonary fibrosis with retraction, intermittent dysphagia |
| 12 | I. 10,000 r<br>40 days<br>II. 7,600 r<br>24 days | 54 | 33 | I. Asymptomatic<br>II. Asymptomatic | I. Bony regeneration<br>II. Vertebral regeneration | I. 12<br>II. 18 | 10 weeks | Herpes zoster after first treatment, died of spinal symptoms |
| 13 | I. 10,000 r<br>41 days<br>II. 9.000 r<br>43 days | 37½ | 31½ | I. Asymptomatic<br>II. Asymptomatic | I. Lung tumor disappeared<br>II. Rib. vertebral recalcification | I. 4<br>II. 24 | 2 | Treatment after 2 resections; posttreatment: neck induration, disturbed arm function<br>Autopsy: No pulmonary tumor, perivertebral involvment, spinal *and* cerebral metastases |

except for one case with mild radiation sickness and another case with moderate radiation sickness where large fields (15 cm ) had to be used both cases responded well to Diamimine

In one case with vertebral erosion herpes zoster developed after radiation corresponding to the irradiated spinal segment

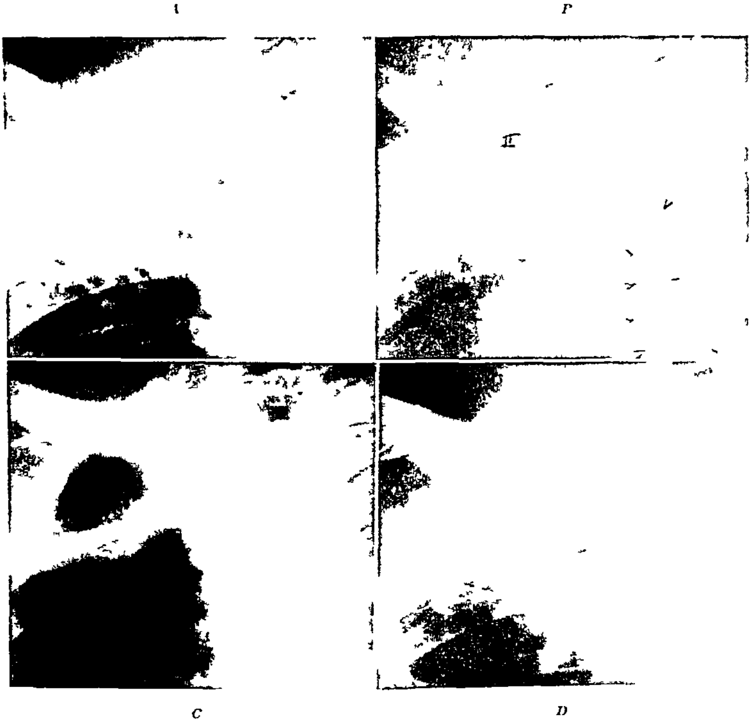


Fig 8—Case 2 A Cavitations in apical tumor Destroyed first and second ribs
B Bucky film. Cross erosions mainly of the third fourth and fifth thoracic vertebrae
C Three months after x ray therapy tumor diminished cavitation penetrate the chest wall
D Eight months after irradiation decreased cavity partially regenerated ribs Retracted upper lobe

More significant were effects of *late damage* These occurred because the patient lived long enough for these to occur and because of the high tumor and tissue dose, which was greater than in the usual series of treatments exceeding the "tolerance" dose especially in patients given a second treatment course

C. *Anatomic Effects.*—

The objective therapeutic effects observed clinically or radiologically were as follows:

1. The supraclavicular neck swelling, due to protrusion of the tumor filled apex, usually started to regress after a few treatments, and disappeared by the time irradiation was completed (see Fig. 7).

2. Metastatic neck nodes usually also regressed.

3. Engorged neck and chest-wall veins, present in one of our cases, rapidly regressed (see Fig. 7).

4. The pulmonary tumor shadow on x-ray films gradually regressed and was replaced slowly by fibrosis, later by homogeneous atelectatic induration (see below). In 2 of our 3 autopsied cases, residual tumor was not found in the lung; and in the third patient, who died shortly after completing radiation, marked local regression was seen, with only small residual tumor nodules remaining in the lung. Occasionally regression of pulmonary cavitation was observed (Fig. 9, *A-C*); however, our clinical results in cases with necrotic tumor cavitations were not favorable.

5. A sensitive objective sign of tumor regression was regeneration of destroyed bones (ribs and vertebrae) (Figs. 6, 10, *A* and *B*), starting a few weeks after irradiation, also shown in figures of our earlier papers.[11, 12] Understandably, vertebral involvement impairs the prognosis. However, it is remarkable that very extensive vertebral destruction also may be favorably influenced by radiation for longer times (Fig. 11, *A-D*). On the contrary, a progressing or newly appearing bony lesion was positive proof of progressive or recurrent disease (Fig. 12, *D*). Partial recalcifications were observed also in such cases in which the tumor regressed in this area, but active lesion was still present in distant parts of the lung or paravertebral area. However, if the regeneration was complete, this occurrence was less probable. A proof of recurrence is also the appearance of tumor mass in the prevertebral soft tissues (Fig. 12, *E*) on lateral or oblique neck films.

6. Regression of the Horner sign was not seen in any of our cases, including those with seemingly healed thoracic lesions, probably because destroyed nerve fibers did not regenerate. Appearance, or increase of Horner syndrome could be observed occasionally during the progression of the disease.

*Radiation reactions and damages:* Some patients had to pay a price for the clinical relief obtained in the form of radiation damage.

Direct radiation reactions were insignificant. The *skin reaction* never exceeded the stage of hyperemia, followed by slight pigmentation or slight scaling which was marked only in cases of short over-all treatment time. In the other cases it was mild.

Direct reactions of subcutaneous tissue were not observed. Complaints related to acute lung or bronchial reactions were not seen. In cases where the mediastinum had to be included in the radiation fields, hoarseness of short duration occurred in one case by tracheal reaction, and mild dysphagia due to esophageal mucositis in 2 cases. Systemic reactions were insignificant,

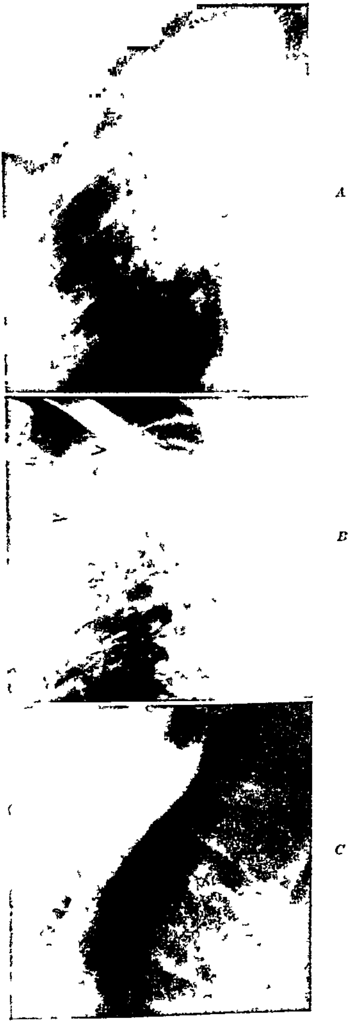


Fig 10—Case 11 *A*, Advanced lung lesion Destroyed second and third ribs *B*, Fourteen months after betatron irradiation regenerated ribs (>) Radiation fibrosis of lung

*C*, Moderate esophageal narrowing in the irradiated area causing intermittent dysphagia.

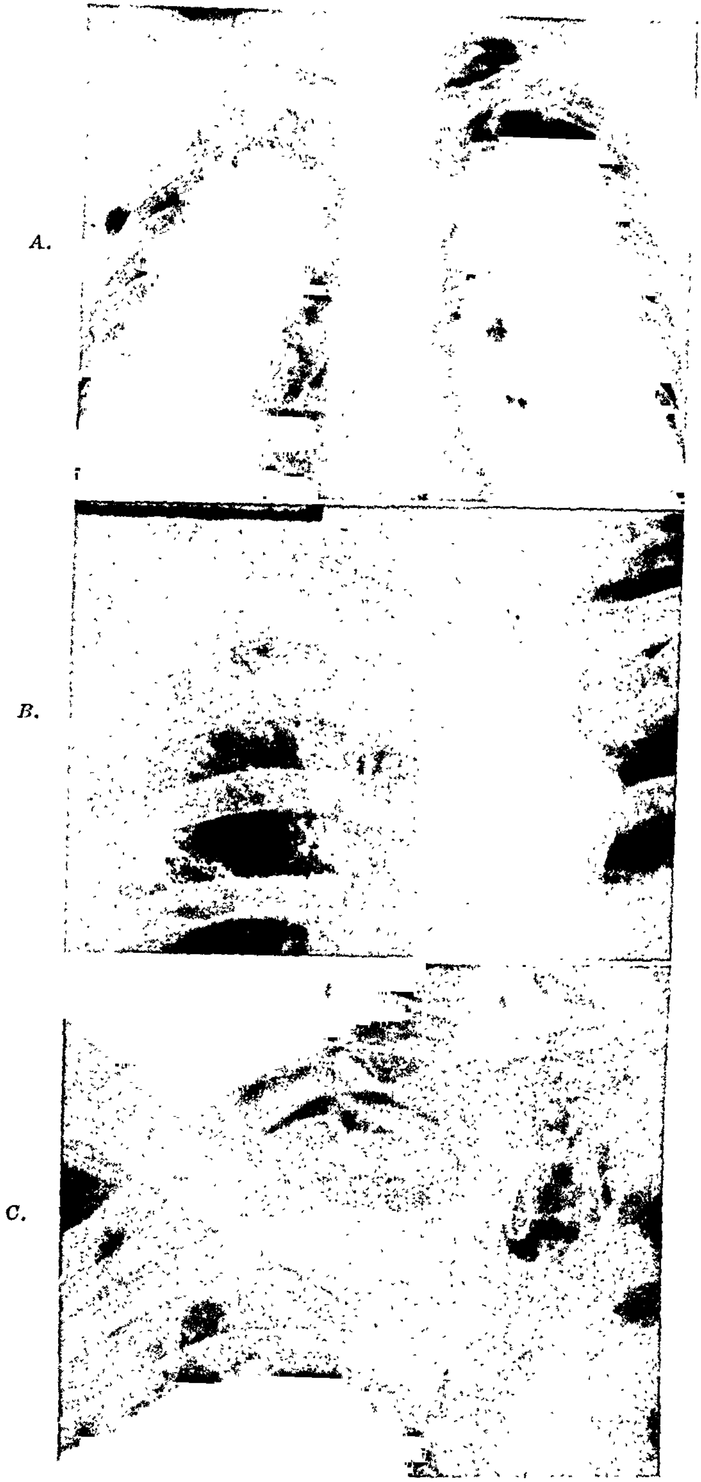


Fig. 9.—Case 16. *A*, Cavitation in right apex. Medially air-bleb. Diagnosed previously as tuberculosis. *Rib lesions shown in Fig. 5.*

*B*, Two months later, before irradiation: cavity enlarged, progression of rib destructions (not shown).

*C*, Six months after irradiation: cavity disappeared. Probably radiation lung fibrosis. Largely recalcified ribs. Recent bony involvement of $T_2$.

Late skin damage consisting of marked atrophy or telangiectasia was not seen In some cases the skin was fixed to the indurated subcutaneous tissues.

Damage of greater clinical significance occurred in the subcutaneous tissues of the supraclavicular area After tumor regression, this fossa was usually rather shrunken, with atrophic muscles due to disuse and to radiation fibrosis After approximately 1½ to 2 years, the fossa filled up gradually by formation of immobile woody-firm scar tissue, fixed to the skin periosteum, giving the appearance of a frozen infiltration (see Biopsies and autopsies showed that this consisted only of fibrotic

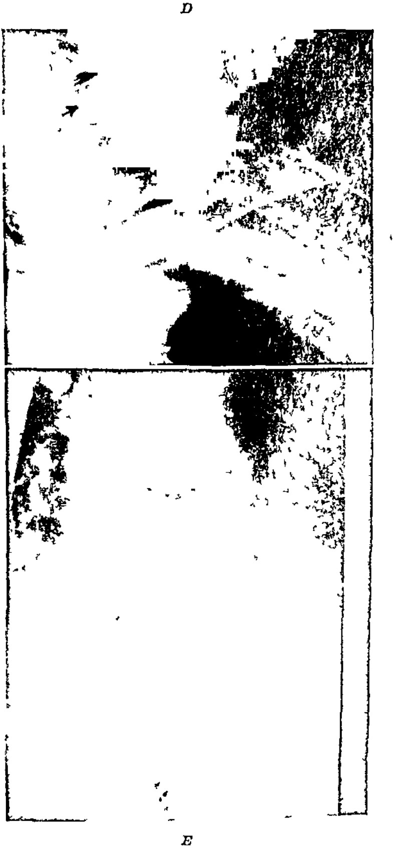


Fig 12—Cont'd *D*, Clinical relapse osteolytic lesion in $T_1$ pedicle area *E*, Mass in prevertebral tissues at the same level (proved by autopsy)

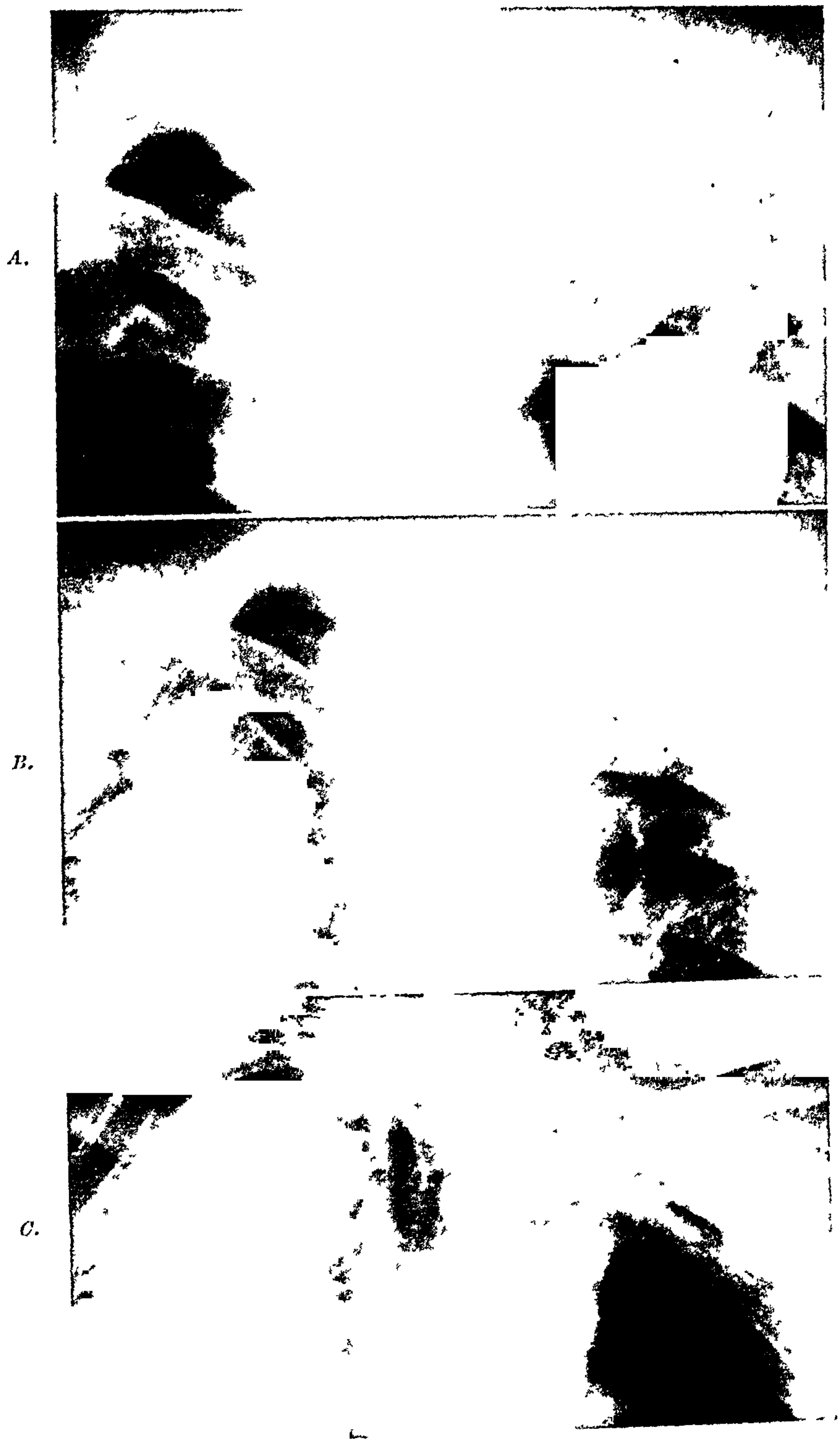


Fig. 12.—Case 13. *A*, Intrapulmonary tumor with chest-wall invasion, proved by resection.

*B*, Recurrence after two surgeries (Erosion of $T_2$ margin, not shown.)

*C*, After two series of betatron irradiation: fibrosis, retraction of irradiated lung. No evidence of bony involvement.

is radioresistant, that radiation offers no benefit at all or only rarely a very slight short-lived relief and diminution of tumor shadow.[5, 8, 10, 16, 1, 22] In some cases, the symptoms were rather aggravated.[6, 16] Only on a few cases was a palliation of several months described,[6, 14, 21] without significant prolongation of life. However, Eschbach and Finsterbusch[6] compiled 71 cases from the literature and 15 cases of their own, and found that, in 37 nonirradiated cases, the average survival from onset was 9.7 months, in 43 irradiated cases, 14 months. From 40 irradiated cases 25 had no effects. In 15 cases improvement was observed, represented in all by pain relief, in 6 by improved general condition, in 4 by decrease of the tumor, and in 2 by recalcification of destroyed ribs. Their data are complicated by inclusion of several cases of nonapical lung tumors, penetrating the chest wall. In recent years our institution has published reports of cases[11, 12] of good radiation effects with regeneration of destroyed ribs, and recently Quick and Richmond[21] also described one case with recalcified ribs.

The comparison of the data presented in our paper with the literature shows that the survival time of our cases treated by conventional x-rays conforms to those cited in the literature, except for one random case cured for 11 years. Of the betatron treated cases, 2 are living and well for 43 and 60 months after the onset, the deceased patients survived the onset by an average of 29.7 months, in Stages 2 to 4 by an average of 43.6 months (literature 12 months). Their average survival after the first irradiation was 19.6 months, in Stages 2 to 4, 32.4 months (literature 6 months[12]). In Stage 5 with metastases, the survival was not prolonged significantly, however a good palliation was achieved in all but one case. Thus radiation results regarding survival and palliation are far better in our betatron group than in our conventional x-ray cases, or than in those described in the literature. We do not believe that the biologic effect of the high energy radiation on the tumor is qualitatively different. The better betatron results should be attributed rather (1) to the higher tumor dose, (2) to the more homogeneous dose distribution in the tumor, and (3) to the more accurate and careful treatment planning and positioning. We believe that if the same care were exerted in treatment planning and technique with conventional x-ray therapy, this also could achieve some degree of results.

As seen, the histopathology of the apical tumors is as diversified as the other bronchogenic cancers, and they do not form a separate pathologic entity.

The response to radiation in Stage 5 is similar to other lung carcinomas but is much better in the earlier stages of the apical tumors. This can be explained by a combination of several factors, related to the peripheral localization and smaller volume of the apex. First, the lesion reaches the pleura and chest wall easier and elicits pain earlier, with a consequently earlier diagnosis. Second, the tumor extends peripherally with greater ease, the central lymphatics are involved later, and the mediastinal nodes less frequently. Therefore, local radiation can be more successful. Finally, the irradiated lung volume is more limited, thus the beams can avoid vital mediastinal organs as well as the opposite lung. Thus, radiation is better tolerated than in other localizations.

This tissue sometimes later surrounded and compressed the brachial vessels and nerve plexus, causing first coolness and other circulatory disturbances in the arm (see Fig. 7, *D*), followed by paresthesias and paresis in the fingers, and limitations in shoulder movement. The discomfort was accompanied by varying degrees of aches usually controlled by varying doses of Empirin. Due to the clumsy use of his arm, one of our patients fractured his upper arm twice in rapid sequence through falls. Disturbed arm function occurred in 3 patients but, in one of these, autopsy disclosed a spinal metastasis as additional cause of the ulnar paralysis. Occasionally narrowing of the prevertebral soft tissues by radiation fibrosis was observed on lateral neck films.

The radiation damage next in importance was that of the *esophageal wall*, with late radiation mucositis and moderate narrowing causing dysphagia. We observed this in 2 cases. The narrowing, confirmed by x-ray examination and by endoscopy, was not significant enough to require dilatation or gastrostomy (see Fig. 10, *C*). In one case, the dysphagia was elicited 3 to 4 times yearly on a psychosomatic basis, and disappeared in 2 to 3 days by change of environment. In the other case, a few weeks after avitaminotic stomatitis (which healed promptly by vitamin medication) apathy and weakness appeared about 4 weeks before his death. One week later dysphagia developed, with the previously mentioned moderate x-ray and endoscopy findings which accounted for reduced food intake. The weakness and lethargic condition progressed until his quiet death, without signs of recurrence or metastases. How much of this malnutrition was due to his alcoholism or to dysphagia could not be analyzed, because he was not hospitalized, but the dysphagia possibly aggravated and accelerated the fate of the patient.

Clinical *bone damage* was not seen, although in one autopsy case the clavicle did show sclerotic changes in the spongiosa.

The irradiated *lung segment* showed radiologically (Figs. 10, *B*, and 12, *C*), and at autopsies in all cases, atelectatic and fibrotic collapse, with shrinking, followed by retraction of the apical thorax and mediastinum. Autopsies showed adhesion of the collapsed segments to the chest wall. The functional loss of the segment caused no clinical symptoms. In some cases moderate local discomfort was the chief complaint, due probably to the adhesions and retraction.

The possibility of *postradiation transverse myelitis* was considered clinically in 2 cases. In one of these, it remained undecided whether the spinal symptoms were produced by radiation myelitis or by neoplastic spinal involvement. However, vertebral bony involvement, clinical similarity to the second case, and inconclusive but suspicious myelogram favor the latter etiology. In the second case, autopsy revealed spinal involvement by tumor, without radiation damage of the spinal cord.

## COMMENT

The literature is very pessimistic about the fate of these patients. The average survival after onset is listed by various authors[17, 22, 23] as 10 to 14 months. According to Moersch and associates,[19] the majority die within 6 months after the first examination. All agree in general that the apical tumor

4 Editorial Cancer Eponyms Pancoast's Tumor, The Cancer Bulletin, Illinois Edition 7, 67, 1955
5 Editorial Superior Sulcus Tumor, Radiology 47 188 189, 1946
6 Eschbach, H, and Finsterbusch, R Die Ausbrecherform des Bronchial Krebses Erg d Inn Med u Kinderhk 65 60 138, 1945
7 Fried, B M Sternoclavicular branchioma, Am J Cancer 25 738 745, 1935
8 Fried, B M Bronchiogenic Cancer Treatment With Roentgen Rays, Am J Cancer 20 791 806, 1934
9 Garland, L H Discussion of Stein's paper[23]
10 Graef, I, and Steinberg, I Superior Pulmonary Sulcus Tumor, Am J Roentgenol 36 293 300, 1936
11 Haas, L L, Harvey, R A, and Langer, S S Radiation Management of Otherwise Hopeless Thoracic Neoplasms, J A M A 154 323 326, 1954
12 Harvey, R A, Haas, L L, and Laughlin, J S Betatron Cancer Therapy, Radiology 58 23 34, 1952
13 Herbut, P A, and Watson, J S Tumor of the Thoracic Inlet Producing the Pancoast Syndrome A Report of 17 Cases and a Review of the Literature, Arch Path 42 88 103, 1946
14 Jacox, H W Superior Pulmonary Sulcus Tumor, J A M A 103 84 87, 1934
15 Jacox, H W, and Baker, M R Primary Apical Lung Cancer Producing the Symptomatology of a Superior Sulcus Tumor, Radiology 29 525 529, 1937
16 Kelman, H, and Schlezinger, N S Tumor of the Superior Thoracic Inlet (Tumor of the Superior Pulmonary Sulcus), Arch Path 23 383 391 1937
17 Leddy, E T Treatment of Intrathoracic Diseases In Portman's Clinical Therapeutic Radiology, New York, 1950, Thos Nelson & Son
18 Liebow, A A Pathology of Carcinoma of the Lung as Related to the Roentgen Shadow, Am J Roentgenol 74 383 401, 1955
19 Moersch, H J Hinshaw, H C, and Wilson, L H Apical Lung Tumors or So Called Superior Pulmonary Sulcus Tumors, Minnesota Med 23 221 226 1940
20 Pancoast, H K Superior Pulmonary Sulcus Tumor, J A M A 99 1391 1396 1932
21 Quick D and Richmond, J D Preliminary Experiences With a 50 Gr Converging Beam Radium Unit, Am J Roentgenol 74 635 649 1955
22 Ray, B S Tumors at the Apex of the Chest, Surg, Gynec & Obst 67 599, 1938
23 Stein, J J Apical Lung Tumors J A M A 111 1612 1617 1938
24 Tobias, J W Sindrome Apico Costo Vertebral Doloroso Per Tumor Apexiano, Rev med latino am 17 1522 1556, 1932

## SUMMARY

1. The apical lung tumors do not form a separate pathologic entity, but a topographic distinction is made because, due to the particular peripheral location, their clinical and radiotherapeutic behavior is different from that of other lung cancers.

2. An attempt is made to subdivide the cases in 5 stages, dependent upon the degree of involvement.

3. Eighteen cases were irradiated at the University of Illinois College of Medicine, 8 with conventional x-rays, 10 with betatron x-rays.

4. The cases are reviewed and analyzed from diagnostic and therapeutic points of view.

5. The betatron radiation excelled by far the conventional x-rays in therapeutic effect, based upon comparisons within our own material as well as with the data in the literature. However, conventional x-ray therapy is also expected to achieve better results if similar care will be exerted in treatment planning and technique.

6. The *conventional* x-ray case figures show one cured for 11 years, regarded as an exceptional curiosity in radiosensitivity. In the other cases, the results conform to the data of the literature.

7. The 10 betatron case figures show 2 living and well, 11 and 27 months after the radiation. The deceased patients survived from the onset an average of 29.7 months, in Stages 2 to 4, 43.6 months (literature: 12 months); from the first irradiation an average of 19.6 months, in Stages 2 to 4, 32.4 months (literature: 6 months). In Stage 5 (with regional or distant metastasis) the prolongation of life was not significant, but the palliative effect was striking, in fact much better than implied in the literature.

8. The radiation effect on subjective symptoms (pain, etc.) was manifest in their disappearance or marked decrease for variable durations, and in significant reduction of the patient's suffering, and duration of the terminal stage.

9. The objective radiation effect could be followed clinically or radiologically by reduction or disappearance of the tumor, by recalcification of destroyed costal or vertebral bony structures, by gain in weight and strength, by resumed and prolonged useful life, and by disappearance or regression of the tumor in autopsied cases.

10. It is worth while to irradiate all cases of apical tumors, in Stages 2 to 4 for significantly prolonged periods of useful life and possible cure, in Stage 5 as a palliative aid to living during the remaining life of the patient. The diagnosis is rarely made in Stage 1, in which surgery would be academically the therapy of choice.

## REFERENCES

1. Barabas, M., and Lendvai, J.: Lungenspitzen Geschwulst. Pancoast Syndrom, Rad. Clin. 19: 36-50, 1950.
2. Browder, J., and DeVeer, I. A.: The Varied Pathologic Basis for the Symptomatology Produced by Tumors in the Region of the Pulmonary Apex and Upper Mediastinum, Am. J. Cancer 24: 506-521, 1935.
3. Clarke, B. E.: Superior Pulmonary Sulcus Tumor (Pancoast), Am. J. Path. 10: 693-696, 1934.

The actual period of by-pass was carried out as follows: The superior vena cava was occluded and its blood siphoned into a graduated cylinder. The left brachial artery was temporarily occluded and the branches of the right also, if it was not used for perfusion. The inferior vena cava was next occluded. The aorta was cross-clamped immediately distal to the origin of the left subclavian artery and perfusion begun (Fig. 1). An incision, 4 to 5 cm. in length, was made in the right ventricle and the coronary sinus blood continuously aspirated (Fig. 2). In all animals, the right ventricle was open for 5½ to 31 minutes. The ventricle was then closed, the lungs were inflated, normal circulation was restored, and perfusion was discontinued.

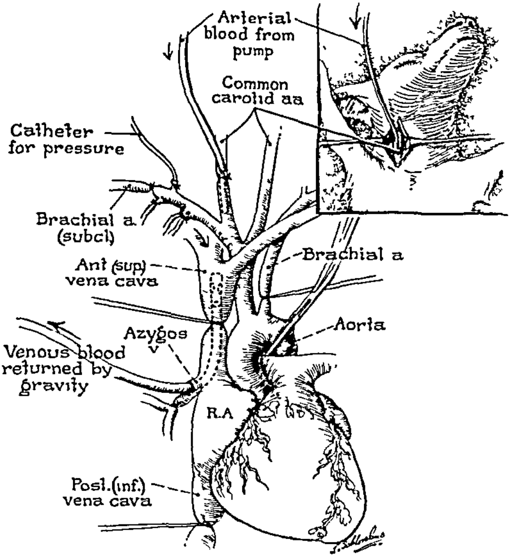


Fig. 1—Schematic representation of the perfusion system

The arterial blood used for perfusion was obtained from the femoral arteries of donor dogs. The blood was collected in bottles containing 40 mg. of heparin per liter of blood. No determinations of blood incompatibility were made and frequently pooled blood from 6 or 7 dogs was used. The blood was pumped from a 2-liter graduate after filtration through commercial recipient sets.

In most animals, the sum of the blood collected from the superior cava and from the right ventricle was 150 to 250 ml. greater than the amount perfused during the period of by-pass. This deficit was replaced slowly with an in-

# CORONARY AND CAROTID ARTERY PERFUSION DURING TOTAL BY-PASS OF THE HEART

JEROME HAROLD KAY, M.D.,* ROBERT A. GAERTNER, M.D., JAMES P. ISAACS,
M.D., AND RICHARD DEVER, M.D.,**
BETHESDA, MD.

As a result of the early and continued interest of Gibbon[6-8] and the recent contributions of Andreasen,[1] Lillehei,[2] and Kirklin,[3] and their associates, repair of ventricular septal defects has become a reality. Recently, Warden and his co-workers[4] reported continuous arterial reservoir perfusion during cardiac by-pass. The following method of perfusing arterialized blood from a reservoir during cardiac by-pass has been used experimentally for the past year and a half. With this technique, only the brain and heart receive blood during the period of by-pass and this paper reports the results of 121 experiments.

## METHOD

Dogs weighing from 4 to 30 Kg. were used. Anesthesia was induced with 2½ per cent Pentothal and an endotracheal tube was inserted. The animals were maintained with ether and oxygen administered with intermittent positive pressure. The femoral artery and vein were cannulated for pressure measurements. A small number of animals were not cooled. The remaining dogs were cooled by immersion in a water and ice bath (3° to 4° C.). The animals were removed from the bath when the rectal temperature was 3° or 4° C. higher than the desired final temperature, to allow for the usual drop. Sterile technique was not employed.

A right thoracotomy was performed through the fourth intercostal space. A heavy silk ligature was passed around each vena cava. The right brachial (subclavian) artery was isolated and ligatures were passed around it and all of its branches, except for the internal mammary, which was cannulated for the measurement of blood pressure during the period of by-pass. The mediastinal pleura was opened and ligatures passed around the left brachial (subclavian) artery and the aorta. A polyethylene catheter was inserted into the right brachial artery in the chest or the right carotid artery in the neck for subsequent perfusion of the aortic arch. This catheter was connected through a finger pump to a reservoir of heparinized, oxygenated blood. Another catheter was inserted into the superior vena cava through the azygos vein.

---

From the Clinic of Surgery, National Heart Institute, Bethesda, Md., and the Department of Surgery of The Johns Hopkins University and Hospital, Baltimore, Md.

Received for publication March 26, 1956.

*Present address: University of Southern California School of Medicine, Department of Surgery, Los Angeles, Calif.

**Present address: University of Miami School of Medicine, Miami, Fla.

GROUP II Eighty-five dogs were cooled to a final rectal temperature of 25° to 30° C The aorta was cross clamped and both brachial arteries were occluded for 15 to 31 minutes The right ventricle was open for 15 to 30 minutes During the period of by pass the blood pressure was maintained at 80 to 90 mm Hg in most of the animals Within a minute after stopping the perfusion and restoring the circulation, the blood pressure was usually above 100 mm Hg Thirty six of the 85 dogs were chronic survivors The remaining 49 died within the first 3 postoperative days Six of the dogs died of cardiac arrest within a few minutes after perfusion and restoration of the circulation In 6, the cause of death could not be determined at autopsy In 10 animals the cause of death was thought to be due to hemorrhage, and in 27 animals the cause of death was pulmonary congestion This was confirmed by pathologic examination

Twenty one of the animals developed ventricular fibrillation during perfusion or after restoration of the circulation It was possible to defibrillate all of the hearts, but only 7 of these 21 dogs were chronic survivors

There were several points of interest in this group of animals A record was kept of the amount of blood recovered from the superior vena cava as compared to the amount recovered from the right ventricle More blood was always recovered from the superior vena cava during the period of by pass but this varied considerably The range was fairly evenly distributed between a ratio of 2 to 8 times as much recovered from the superior vena cava as from the right ventricle In a few animals, 14 and even 18 times as much blood was perfused to the head as to the heart There was no relationship between this ratio and survival

In this group of animals, the amount of blood required to maintain a pressure of 80 to 100 mm Hg during the period of by pass was about 150 c c per minute in a 15 Kg dog There was a great deal of individual variation, however

In 9 animals, immediately after opening the right ventricle, a Foley bag catheter was inserted into the pulmonary artery and the bag inflated Through this catheter, blood was perfused into the lungs by gravity during the period of by pass Ten to 35 c c of blood were perfused per minute in a 10 to 15 Kg dog The survival rate in this group was no better than when only the head and heart were perfused, 5 of these 9 dogs died

Twenty animals were digitalized prior to cardiac by pass Seven of the animals were digitalized by giving them 0 15 mg of acetyl strophanthidin in three doses The drug was given intravenously at 5 minute intervals so that the last injection was given just prior to by pass Nine of the dogs were given 1 mg of Digoxin intravenously 4 hours prior to cooling and another 0 5 mg intravenously as the chest was being opened The initial dose always made the dogs vomit Of these 20 dogs, only 8 were chronic survivors, so that there was no increased survival among the dogs that received a digitalis preparation

In 34 animals, the lungs were oxygenated with 100 per cent oxygen during the period of by pass Ether was not administered during this period Of these travenous transfusion of citrated blood after restoration of the circulation The animals were given protamine sulfate (1 mg /Kg.) to counteract the heparin that remained following perfusion

## RESULTS

Group I. This technique was used to by-pass the heart in 11 dogs under normothermic conditions. The aorta and brachial arteries were occluded for 8 to 18 minutes in these animals, and the right ventricle was open for 5½ to 17 minutes. Four of the animals were chronic survivors. The period of occlusion in these surviving animals was 8½, 12, 14, and 18 minutes Four of the dogs died 16 to 36 hours after surgery. The aorta and brachial arteries were clamped for 12 to 15 minutes, and all 4 dogs revealed evidence of cord damage with spastic paralysis of the hind legs Three more animals died of postoperative bleeding within the first 24 hours

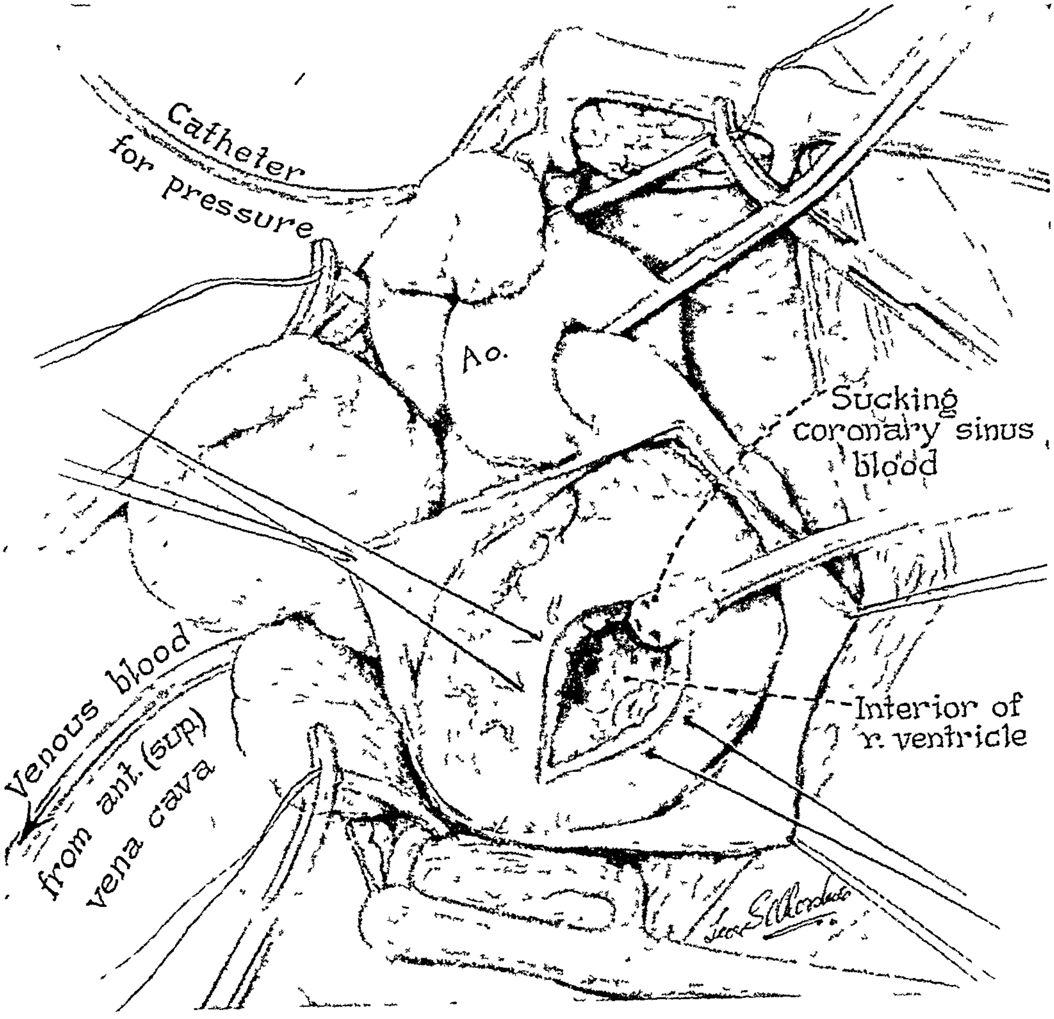

Catheter for pressure
Ao.
Sucking coronary sinus blood
Interior of r. ventricle
Venous blood from ant.(sup) vena cava

Fig. 2—Operative exposure obtained during the period of complete by-pass

by pass None of these factors seemed to influence the survival rate Several factors seemed to be important The final rectal temperature should not be lower than 30° to 32° C In this range there was no ventricular fibrillation whereas in the animals perfused at a final rectal temperature of 25° to 30° C there was a 25 per cent incidence of ventricular fibrillation

It was important to maintain a blood pressure of at least 85 mm Hg during the period of by pass Slow release of the inflow clamps over a period of 4 to 5 minutes and release of the aortic clamp over a period of ½ minute seemed important in order to maintain a good blood pressure and coronary circulation Despite all of these precautions, however, 4 of the last 25 dogs died with pronounced pulmonary congestion within 48 hours after operation In all of these animals, the right ventricle was open for 15 to 30 minutes These results are similar to those reported by Shumway, Gliedman, and Lewis[5] They perfused the coronary but not the carotid vessels after first cooling the animals to 25° C They felt that in dogs it was safe to by pass the heart for periods up to 20 minutes at this temperature with coronary perfusion of 50 to 85 c c per minute

## SUMMARY

A technique is described for cardiac by pass in the dog permitting right ventriculotomy for 30 minutes The head and heart are perfused with oxygenated, heparinized blood while the animal is maintained at a rectal temperature of 30° to 32° C In the last group of 25 dogs, 21 are long term survivors

## REFERENCES

1 Andreasen A T, and Watson F Experimental Cardiovascular Surgery Discussion of Results So Far Obtained and Report on Experiments Concerning a Donor Circulation Brit J Surg 41 195, 1953
2 Lillehei C W, Cohen M, Warden H E, and Varco R L The Direct Vision Intracardiac Correction of Congenital Anomalies by Controlled Cross Circulation Results in 32 Patients With Ventricular Septal Defects Tetralogy of Fallot and Atrioventricularis Communis Defects Surgery 38 11 1955
3 Kirklin J W DuShane J W Patrick R T Donald D E Hetzel P S Harshbarger H G, and Wood E H Intracardiac Surgery With the Aid of a Mechanical Pump Oxygenator System (Gibbon type) Report of Eight Cases Proc Staff Meet, Mayo Clin 30 201, 1955
4 Warden, H E, Read R C DeWall R A, Aust J B Cohen M Ziegler N R Varco, R L, and Lillehei, C W Direct Vision Intracardiac Surgery by Means of a Reservoir of "Arterialized Venous" Blood Description of a Simple Method and Report of the First Clinical Case J THORACIC SURG 30 649 1955
5 Shumway, N E, Gliedman, M L, and Lewis, F J *Coronary Perfusion for Longer Periods of Cardiac Occlusion Under Hypothermia J THORACIC SURG 30 598 1955*
6 Gibbon, J H, Jr Artificial Maintenance of Circulation During Experimental Occlusion of Pulmonary Artery A M A Arch Surg 34 1105, 1937
7 Stokes, T L, and Gibbon, J H Jr Experimental Maintenance of Life by a Mechanical Heart and Lung During Occlusion of the Venae Cavae Followed by Survival, Surg, Gynec & Obst 91 138, 1950
8 Gibbon J H, Jr, Miller, B J Dobell A R, Engell H C and Voigt G B The Closure of Interventricular Septal Defects in Dogs During Open Cardiotomy With the Maintenance of the Cardiorespiratory Functions by a Pump Oxygenator, J THORACIC SURG 28 235, 1954

34 animals, 15 were long-term survivors. Therefore, the mortality in this group was about the same as the over-all mortality.

GROUP III. Twenty-five dogs were cooled to a final rectal temperature of 30° to 32° C. The aorta was clamped for 15 to 37 minutes, and the right ventricle was open for 15 to 31 minutes. In this group of animals, an attempt was made to apply the information gained in previous experiments. The lungs were deflated during the period of by-pass in this group. A blood pressure of 85 to 125 mm. Hg was maintained in all of these dogs during the period of by-pass. In this series of experiments, a 15 Kg. dog received approximately 175 c.c. of blood per minute to maintain this pressure during the period of by-pass. One minute after closing the ventricle the tie around the superior vena cava was released, allowing some of the superior vena caval blood to enter the right atrium. Over the period of the next minute to a minute and one-half, the catheter in the superior vena cava was gradually occluded, so that more blood returned to the right atrium. The ties around both brachial arteries were released. The pump was gradually slowed and the inferior caval constriction gradually released. Extreme care was taken to prevent overdistention of the right atrium. The aortic clamp was gradually released. None of the animals developed ventricular fibrillation during the period of by-pass or after restoration of the circulation. Four of the dogs died within 36 hours of pulmonary congestion, and the other 21 are long-term survivors.

## DISCUSSION

Prior to using the method just described, total by-pass of the heart was complished by using a dispersion type oxygenator and a finger pump. In 7 animals, the entire animal was perfused and, in 14 more, the aorta was cross-amped at the origin of the left brachial artery during the 15 to 30 minutes by-pass and right ventriculotomy. A systolic pressure of 80 mm. Hg was sually maintained during this period. All of the dogs except one died of ock, bleeding, or pulmonary congestion.

Because of these discouraging results, it was felt that the survival rate uld be improved if the heart and head alone were perfused. In this way, the ol of normal blood in the rest of the body would not enter the extracorporeal rcuit. In the early experiments with carotid and coronary artery perfusion, e body temperature was not lowered. However, with the aorta and both achial (subclavian) arteries clamped, cord damage frequently occurred with riods of occlusion of 12 to 15 minutes. Hypothermia was then employed, with ctal temperatures of 25° to 30° C., but usually closer to 25° than to 30° C. owever, because of an incidence of ventricular fibrillation of 25 per cent, and cause of a chronic survival rate of only 40 per cent, the animals were per-used at progressively higher temperatures.

In the course of the experiments, an attempt was made to evaluate certain factors. In some of the animals, the pulmonary artery was perfused with blood during the period of by-pass. Another group of animals was digitalized. In still another group of animals, the lungs were inflated during the period of

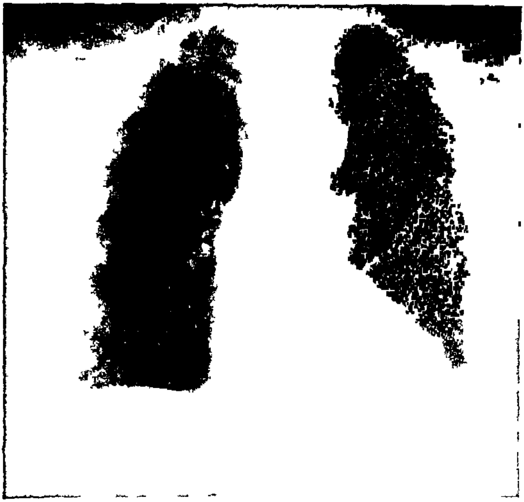


Fig 1—Roentgenogram of the chest demonstrates a fluid level in the intrathoracic portion of the stomach

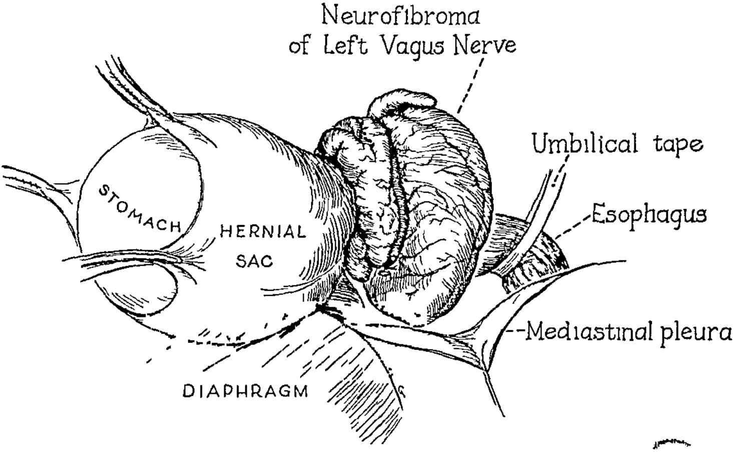


Fig 2—Drawing sketched at operation shows the relationship of the neurof
esophageal hiatus hernia

# INTRATHORACIC NEUROFIBROMA OF THE VAGUS NERVE ASSOCIATED WITH A DIAPHRAGMATIC HERNIA

Carl Davis, Jr., M.D., and Gordon Brown, M.D.
Chicago, Ill.

A MOST unusual and completely unsuspected finding during the course of the repair of an esophageal hiatus hernia of the diaphragm was a large neurofibroma of the vagus nerve supplying the lower end of the esophagus.

In a review of their own series of 18 patients with intrathoracic neurofibroma and in 100 cases of others collected from the literature, Kent, Blades, Valle, and Graham[1] found no evidence of the vagus nerve as a point of origin of these nerve tumors. The infrequency of reports of neurofibromas of the intrathoracic as compared to the cervical portion of the vagus nerve suggests the rarity of this lesion. We have encountered only four such specific reports in the available literature. In 1943, Blades and Dugan[2] reported a case of multiple intrathoracic nerve fibromas, two of these originating in the left vagus nerve. In 1950, Parrella[3] reported a neurofibrosarcoma of the left vagus nerve which was located at the base of the neck and extended into the anterior superior mediastinum. In 1953, Gerbode and Marguiles[4] reported an interesting case of neurofibromatosis (von Recklinghausen's disease) with neurofibromas involving the left vagus nerve in the superior mediastinum. A year later, Gilbertsen and Lillehei[5] published a case report which concerned a 40-year-old man with bilateral cervicothoracic neurofibromas of the vagus nerves in the absence of neurofibromatosis. Physiologic observations were carried out to study the effect of removal of the tumors on the function of the cardiorespiratory systems. The usual site of intrathoracic nerve sheath tumors is in the posterior mediastinum with origin from the sympathetic chain or the intercostal nerves, rather than the vagus nerve.

## CASE REPORT

Mrs. J. K. was first seen in 1944 when, at the age of 55, she was referred by her family physician with the diagnosis of pernicious anemia refractory to liver therapy. Signs and symptoms of anemia had then been present for one year and she had had five blood transfusions. Initial investigation, in 1944, revealed a normocytic hypochromic anemia, occult blood in the stools, normal gastric secretions, and an esophageal hiatus hernia (Figs. 1 and 2). On the diagnostic assumption that bleeding resulted from peptic erosion associated with the diaphragmatic hernia, treatment comprised modified Sippy management plus transfusions and iron. Although the therapeutic response was prompt, the patient refused to remain on medical measures after the acute symptoms subsided. During the following twelve years she had 15 major gastric hemorrhages. From 1947 on, operative repair of the hiatus hernia was recommended with increasing persistence because of the conviction that

---

From the Departments of Surgery and Internal Medicine, The Presbyterian Hospital, Chicago, Ill., and The University of Illinois College of Medicine (Rush), Chicago, Ill.

Received for publication March 29, 1956.

The patient was re-examined six weeks after operation. The wound was well healed. There were no particular complaints. Solid foods were swallowed without difficulty. At this time a roentgen examination of the esophagus and cardiac end of the stomach showed no evidence of stenosis, regurgitation, or ulceration. The stomach was confined to the peritoneal cavity.

*Pathology—gross examination:* The mediastinal tumor consisted of a somewhat circular mass of gray tissue, measuring 9.5 by 4.5 by 2.3 cm (Fig. 3). The specimen weighed 60 Gm. On one side of the specimen there was a triangular section of gastric mucosa removed from the cardiac end of the stomach. On the interior side of the specimen there was a portion of the left vagus nerve, measuring approximately 2.5 cm. in length. At one point there was evidence of bifurcation of the nerve.

*Pathology—microscopic examination:* The tumor was fibrous in character and consisted essentially of a proliferation of Schwannian cells There was massive fibrous overgrowth with interspersed hyaline fibers, often arranged in concentric groups between interlacing bundles of elongated cells, having many of the features of fibrocytes The tumor was relatively acellular in various places, the result of abundant collagenous deposits The palisade arrangement of nuclei was not as conspicuous as in some neurogenic tumors (Fig. 4) In one section, there were several areas of calcification; in another section, at the level of the esophageal gastric junction, an intense subacute inflammation underlying the stratified squamous epithelium was present; however, the gastric mucosa appeared normal The histologic diagnosis was neurofibroma of the vagus nerve, subacute inflammation of the esophagus

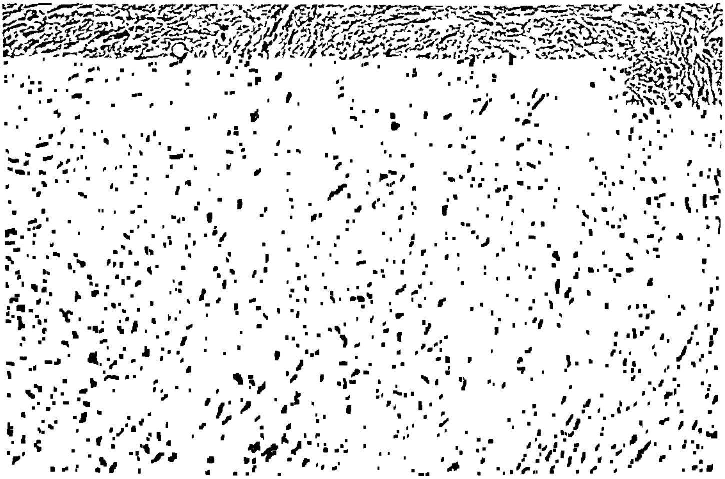


Fig. 4—Photomicrograph of the neurofibroma showing the nuclei of nerve cells disseminated through interlacing bundles of fibrous connective tissue

## DISCUSSION

Of salient interest in this case is the 12-year history of recurrent gastric hemorrhages, associated with a large esophageal hiatus [illegible] ready response of these bleeding episodes to acid-neutralizing [illegible] lief peptic ulceration was occurring in this pouch although an ulcer crater could not be conclusively demonstrated fluoroscopically. Finally, after a particularly depleting episode of melena had been brought under control, the patient entered the Presbyterian Hospital, Chicago, on Aug. 31, 1955, for surgical treatment of a diaphragmatic hernia. It is of particular interest that the patient had never experienced abdominal or thoracic discomfort and had no digestive tract symptoms or signs other than those due to hemorrhage. There were no pregnancies. There was no history of trauma. Extreme obesity had been present for thirty years.

Examination on admission revealed a 66-year-old white woman, 65 inches in height, weighing 200 pounds. The blood pressure was 198/100 mm. Hg. Other physical findings were normal, as were the hemogram, urinalysis, electrocardiogram, and examination of the feces.

On Sept. 1, 1955, a left thoracotomy was performed through the bed of the resected seventh rib. Upon incising the mediastinal pleura, the preoperatively diagnosed esophageal hiatus hernia presented itself, as well as an unsuspected gray white, semilunar-shaped tumor which encircled the lower end of the esophagus. The origin of the tumor was a large trunk of the left vagus nerve. A frozen section identified this lesion as a neurofibroma.

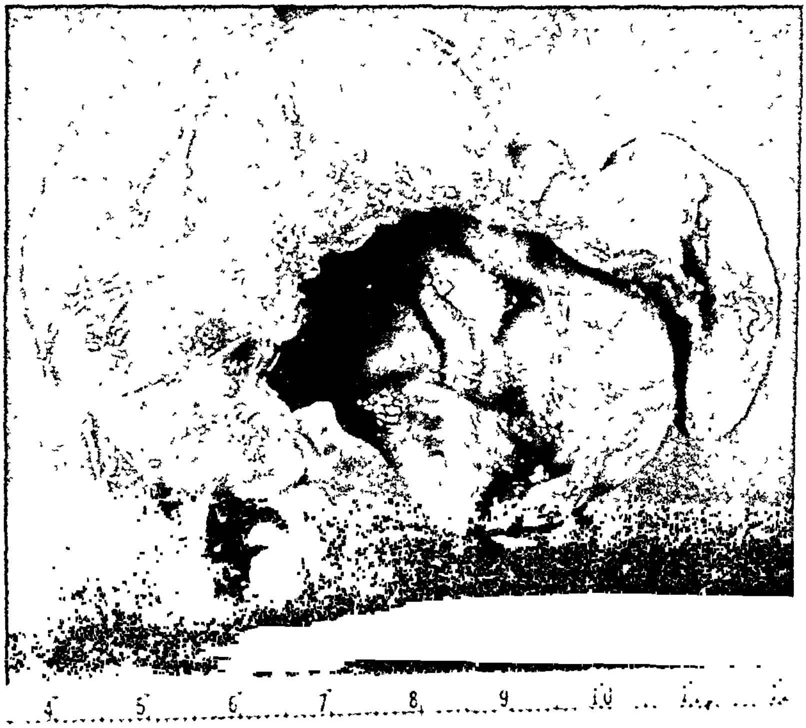


Fig. 3.—Photograph of the surgical specimen.

While resecting the tumor, a small segment of the anterior wall of the stomach was removed at the level of the cardio-esophageal junction. Exploration through the opening in the gastric pouch revealed no evidence of recent or old peptic ulceration nor any esophageal varices. In closure of the defect, an anastomosis was accomplished between the anterolateral wall of the esophagus and adjacent stomach. The excess portion of the hernial sac was excised. The esophageal hiatus was reconstructed with transplantation of the esophagus, anteriorly. After an uneventful postoperative course, the patient was discharged on the sixteenth postoperative day.

# VENTRICULAR FIBRILLATION

## AN EXPERIMENTAL STUDY COMPARING VARIOUS VOLTAGES AND DURATIONS OF ELECTRIC SHOCK IN DEFIBRILLATION OF THE CANINE HEART

GEORGE C. KAISER, M.D.,* JOHN H. EDGCOMB, M.D., AND
JEROME HAROLD KAY, M.D.**
BETHESDA, MD.

THE use of electric shock is the accepted method for the treatment of ventricular fibrillation. The design of the electrical defibrillator is generally agreed upon but there is still disagreement as to the optimal voltage and duration of the shock. Kirby, Johnson, Engelberg, and Rovis[1] concluded that 220 volts for 1/10 second was uniformly more successful than lower voltages in defibrillating canine hearts. They felt that this voltage and duration of shock would not cause burns of the heart. Kay[2] and Mackay, Mooslin and Leeds[3] have recommended lower voltages. Because of these conflicting results the following study was undertaken.

### METHOD

Forty-eight mongrel dogs weighing 7 to 15 Kg. were anesthetized with intravenous thiopental sodium. An endotracheal tube was inserted and 100 per cent oxygen was administered by intermittent positive pressure through a demand valve. Using sterile technique, a left thoracotomy was performed through the fifth intercostal space and the pericardium was opened widely anterior to the phrenic nerve. Ventricular fibrillation was induced by passing a shock of 10 volts for ½ to ¾ second through the heart. The heart was allowed to fibrillate for four minutes during which time ventilation was continued. The heart was then massaged for two minutes at a rate of 40 to 50 times per minute and a single shock was then used in an attempt to defibrillate the heart. After defibrillation, the pericardium was left open and the chest was closed in layers. The dogs were sacrificed at intervals of two to twenty-six days and the hearts were examined.

The dogs were divided into four groups of twelve. In all four groups the heart was massaged for two minutes after the four-minute period of fibrillation. In the first group, defibrillation was attempted with a single shock of 130 volts at 1/10 second duration. In the second group, defibrillation was attempted with a single shock of 130 volts at ¼ second duration. The third group was treated with a single shock of 230 volts at 1/10 second. In the fourth group

---

From the Clinic of Surgery, National Heart Institute, and the Pathological Anatomy Branch, Clinical Center, National Institutes of Health, Bethesda 14, Md.

Received for publication April 6, 1956.

*Present address: Department of Surgery, Indiana University School of Medicine, Indianapolis, Ind.

**Present address: Department of Surgery, University of South [illegible] forn [illegible] of Medicine, Los Angeles, Calif.

that evidences of peptic ulceration would be found at operation. The absence of such evidence or signs of venous thrombosis in the gastric pouch eliminates the mechanisms of hemorrhage considered most likely in this case. The inadequacy of present knowledge concerning this important complication, which has an incidence as high as 20 per cent among symptom-producing hiatus hernias, is further emphasized.[5]

Although symptoms from this hernia appeared at an age common for such lesions and in a very obese woman, no definite etiologic factors can be implicated. In spite of the intimate association of the vagus neurofibroma with the hernia, it seems unlikely that either one contributed to the development of the other. The finding of calcium in the tumor suggests the chronicity of this lesion and the 12-year presence of the hernia is a matter of radiologic record.

The operative procedure has produced an apparent cure. No deleterious effects have been observed to result from the resection of portions of the left vagus nerve and the neurofibroma.

Although there are a number of reports of nerve sheath tumors arising from the vagus nerve in the neck, this case is unusual in that only four other instances of intrathoracic tumors arising from the vagus nerve are found in the available literature.

## CONCLUSIONS

The case is that of an elderly, obese, white woman in whom a large esophageal hiatus hernia had been repeatedly demonstrated for twelve years. Her symptoms over this long period were those of acute and chronic blood loss, presumably due to peptic erosion in the gastric pouch. Although numerous attempts had been made to bring the patient to surgery, it was only a particularly massive gastric hemorrhage that prompted the patient to consent to operative treatment.

At operation, a mediastinal tumor was found with its origin in the left vagus nerve innervating the lower end of the esophagus. Histologically, this resembled a neurofibroma. Repair of the esophageal hiatus hernia resulted in disappearance of symptoms. At the present time, seven months postoperatively, there are no complaints referrable to the surgery. The patient is swallowing solid foods as well as liquids without any difficulty. The anemia is corrected.

## REFERENCES

1. Kent, E. M., Blades, B., Valle, A. R., and Graham, E. A.: Intrathoracic Neurogenic Tumors, J. THORACIC SURG. 13: 116, 1944.
2. Blades, B., and Dugan, D. J.: Resection of the Left Vagus Nerve for Multiple Intrathoracic Neurofibromas, J. A. M. A. 123: 409, 1943.
3. Parrella, G. S.: Neurofibrosarcoma of the Vagus Nerve, New England J. Med. 242: 324, 1950.
4. Gerbode, F., and Marguiles, G. S.: Neurofibromatosis With Intrathoracic Neurofibromas of Vagus Nerve, J. THORACIC SURG. 25: 429, 1953.
5. Gilbertsen, V. A., and Lillehei, C. W.: Bilateral Intrathoracic Neurofibromas of the Vagus Nerves, J. THORACIC SURG. 28: 78, 1954.
6. Sweet, R. H.: Analysis of 130 Cases of Hiatus Hernia Treated Surgically, J. A. M. A. 151: 376, 1953.

### PATHOLOGIC OBSERVATIONS

*Gross Findings.*—Thirty seven of the 38 hearts that were defibrillated with one shock were examined grossly and microscopically. They were weighed with the blood removed and a minimum of the great vessels remaining. The weights were between 61 and 125 Gm. Some of the hearts revealed minimal subepicardial hemorrhage that was attributed to massage.

There were no grossly visible burns of the hearts that were defibrillated with 130 volts at either 1/10 or ¼ second. Of the hearts defibrillated with 230 volts at 1/10 second, three of four burns were grossly visible, and of those hearts defibrillated with 230 volts at ¼ second, six of nine burns were visible grossly. The lesions were located on the right ventricle just below the right atrial appendage and at the apex of the left ventricle. These were the sites of application of the electrodes.

*Microscopic Findings.*—Two sections, stained with hematoxylin and eosin were prepared from each of 37 hearts defibrillated with a single shock. The sections of the heart from the thirty eighth dog, defibrillated with 230 volts at 1/10 second, were lost. The blocks were taken to include the entire ventricular wall at the point of contact of the electrodes.

The burns were divided into three groups according to the following criteria. The minimal lesions were less than one low power field (10×) and located in either the outer or middle one third of the myocardium. The moderate lesions were larger than one low power field and located in the outer one third of myocardium, or smaller than one low power field and located in the inner one third of the myocardium. If the lesions were greater than one low power field and located in either the middle or inner one third of the ventricular wall, they were classified as severe.

Two of the 8 hearts defibrillated with 130 volts at 1/10 second revealed microscopic burns. These were extremely small and peripheral in location. They were classified as minimal (Fig. 1*C*). Three of the 10 hearts defibrillated with 130 volts at ¼ second had burns that were moderate, and one other was minimal. Of the 10 hearts defibrillated with 230 volts at 1/10 second, 9 were examined, revealing four burns. There was an extensive severe burn involving the entire thickness of the right ventricle of one heart (Fig. 1*A*). Of the remaining lesions, two were moderate and one was minimal (Figs. 1, *D* and *E*). In the series of hearts defibrillated with 230 volts applied for ¼ second, 9 of 10 hearts showed myocardial burns of varying degree. Two lesions were classified as severe (Fig. 1*B*), two as moderate, and five as minimal (Fig. 1*F*).

### DISCUSSION

Hooker, Kouwenhoven, and Langworthy[2] demonstrated that a current of less than 0.8 ampere will not stop fibrillation and a current greater than 0.8 ampere will not induce fibrillation. Mackay, Mooslin, and Leeds[3] reported that a current greater than 1.5 amperes stopped fibrillation and in no of animals, 230 volts at ¼ second was the initial shock. The failures in all groups were massaged for one minute and defibrillated wtih a single shock of either 230 volts at 1/10 second or 130 volts at ¼ second. Only the hearts of those dogs that had been defibrillated on the first attempt were examined for evidence of myocardial burns. Microscopic sections were taken from the sites where the paddles were applied.

The electrical defibrillator was that described by Kouwenhoven and Kay[4] with the addition of a 1-2 step-up isolation transformer and an electronic timing unit.[*] The timing unit permitted shocks of 1/10 and ¼ second with an accuracy of 1/100 second. The electrodes were made of brass rods, 6 mm. in diameter, with one end bent at a right angle to the axis of the rod. These rods were insulated with rubber tubing. A stainless steel disk, 45 mm. in diameter, was riveted to the bent end of each electrode. Plaster felt, 4 m.m. thick, was sewn to the disks. The disks were saturated with normal saline before being applied to the heart.

## RESULTS

In the first group, a single shock of 130 volts for 1/10 second was effective in 8 of 12 animals (Table I). Ten of 12 animals, comprising the second group, were defibrillated with 130 volts for ¼ second. In the third group, 10 of 12 animals were defibrillated using 230 volts for 1/10 second. Two hundred and thirty volts for ¼ second was successful in 10 of 12 animals in the fourth group. A second shock of either 130 volts for ¼ second or 230 volts for 1/10 second was effective in the total of 10 dogs of the four groups in which fibrillation persisted after the first shock. Serial shocks were not employed in this study. Eight of the 48 animals died. Six of the 8 died within three days and were found to have bilateral pulmonary congestion. The remaining 2 animals died on the third and seventh postoperative days with overt evidence of brain damage. Their lungs were normal.

TABLE I

| INITIAL SHOCK | TOTAL NUMBER OF DOGS | NUMBER OF HEARTS DEFIBRILLATED WITH INITIAL SHOCK | NUMBER OF MYOCARDIAL BURNS | | | |
|---|---|---|---|---|---|---|
| | | | MINIMAL | MODERATE | SEVERE | TOTAL |
| 130 V *1/10 second* | 12 | 8 | 2 | 0 | 0 | 2 |
| 130 V ¼ second | 12 | 10 | 1 | 3 | 0 | 4 |
| 230 V 1/10 second | 12 | 10[*] | 1 | 2 | 1 | 4 |
| 230 V ¼ second | 12 | 10 | 5 | 2 | 2 | 9 |

[*] Sections of one heart were lost. Nine hearts were examined microscopically.

Of the 8 animals that died, 3 had required a second shock for defibrillation. These 3 animals died of pulmonary congestion and did not exhibit evidence of brain damage.

---

[*] Designed and constructed by the Instrument Section, National Institutes of Health, Bethesda, Md.

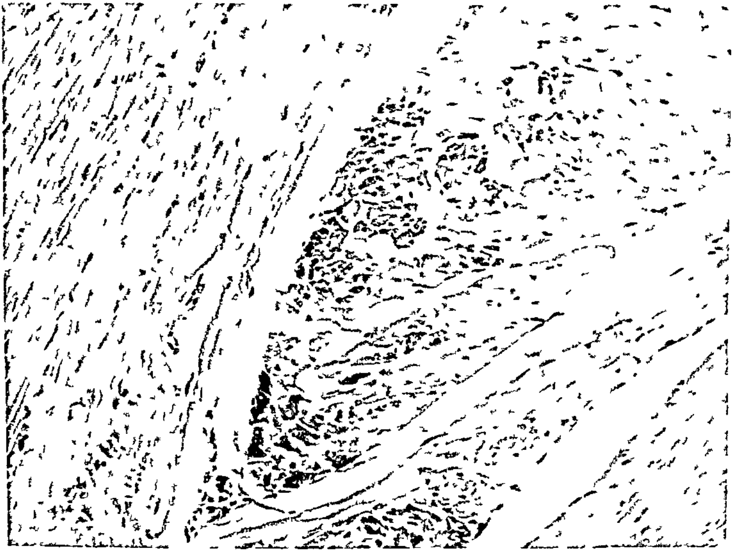


Fig 1C—Minimal focal necrosis of the myocardium of the left ventricle eight days postoperatively The heart was defibrillated with a shock of 130 volts at 1/10 second (×200)

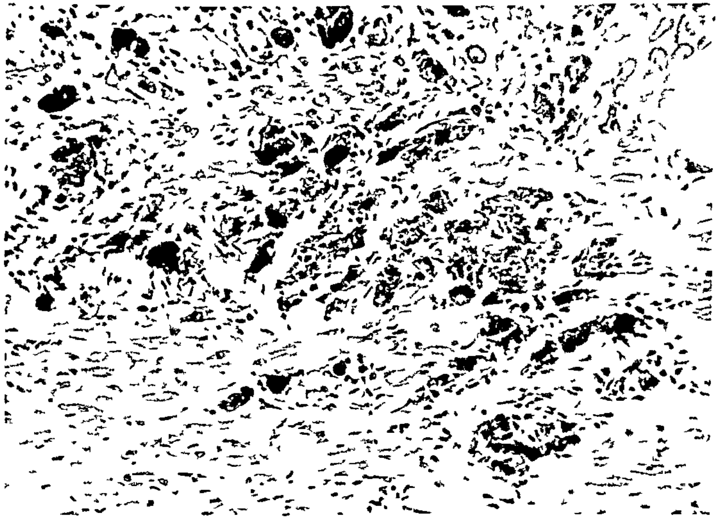


Fig 1D—Minimal lysis necrosis and fibrosis of the myocardium of the right ventricle twelve days postoperatively The heart was defibrillated with a shock of [illegible]30 volts at 1/1 second (×200)

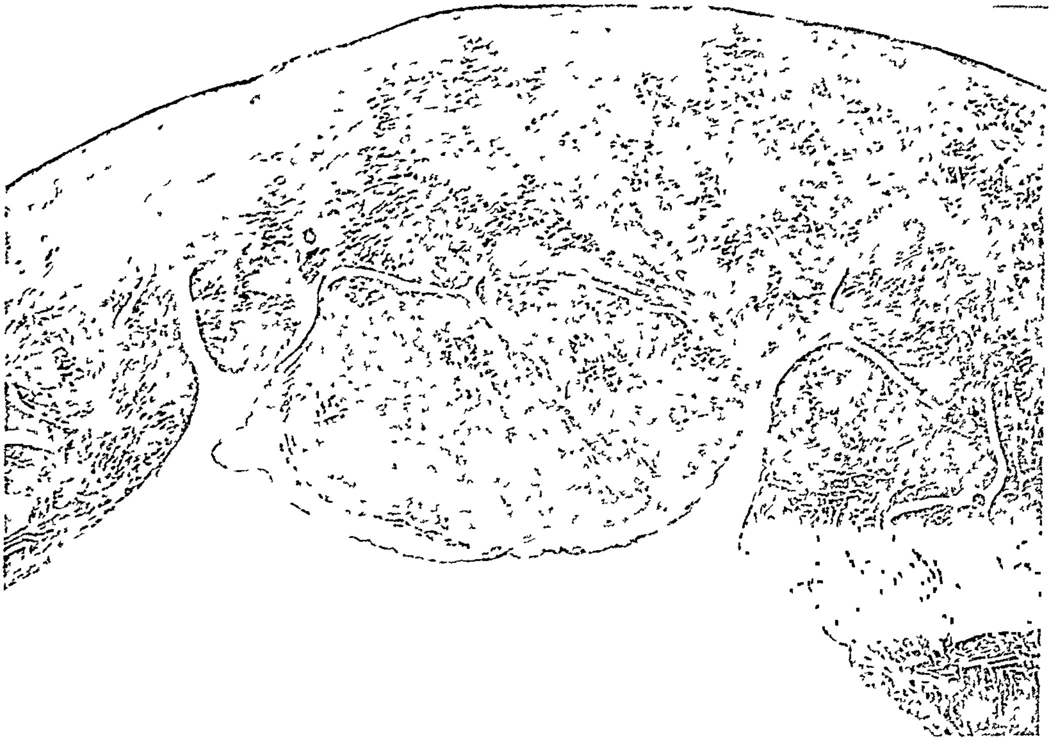


Fig. 1*A*—Extensive necrosis and fibrosis of the right ventricle nine days postoperatively The heart was defibrillated with a single shock of 230 volts at 1/10 second (×13)

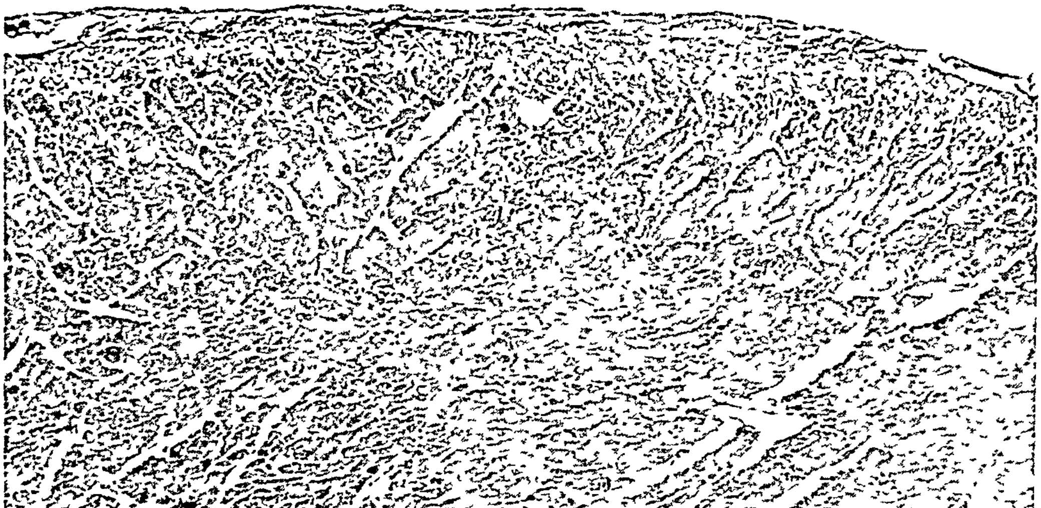


Fig 1*B*.—Extensive necrosis and fibrosis of the myocardium of the left ventricle eight days postoperatively. The heart was defibrillated with a single shock of 230 volts at ¼ second (×58)

produced it They concluded that currents of high amperage may be of either short or long duration to stop ventricular fibrillation and recommended 110 volts at 1/10 second

Kirby, Johnson, Engelberg, and Rovis[1] compared the effectiveness of various voltages at 1/10 second duration They stated that 220 volts at 1/10 second was uniformly more successful than lower voltages at 1/10 second in defibrillating canine hearts They reported that this voltage at 1/10 second duration did not cause burns of the heart, but if the shock lasted ½ second there was a grossly visible burn In the present series voltages and duration of shock were compared Statistically there did not appear to be any significant difference in the ability to defibrillate the heart using the various voltages at the durations studied There were myocardial burns in all four groups However, there was an increased incidence of burns of the heart using 230 volts at ¼ second compared to the other groups The burns of the hearts defibrillated with 130 volts were small and not visible grossly The only severe burns occurred in the hearts defibrillated with 230 volts

## SUMMARY

Ventricular fibrillation was produced in 48 dogs The hearts were allowed to fibrillate for four minutes and cardiac massage was then employed for two minutes An attempt was then made to defibrillate the hearts with single electric shocks of various voltages and durations There did not appear to be a significant difference in the ability to defibrillate the heart using 130 volts at 1/10 or ¼ second duration compared to 230 volts at 1/10 or ¼ second duration However, there was a significant increase in number and severity of burns with 230 volts at ¼ second

## REFERENCES

1 Kirby, C K, Johnson, J, Engelberg, J, and Rovis R Ventricular Fibrillation An Experimental Study, Surg Forum (1953) 4 100 103 1954
2 Kay, J H The Treatment of Cardiac Arrest An Experimental Study, Surg, Gynec & Obst 93 682 690, 1951
3 Mackay, R S, Mooslin K E, and Leeds S F The Effects of Electric Currents on the Canine Heart With Particular Reference to Ventricular Fibrillation, Ann Surg 134 173 185, 1951
4 Kouwenhoven W B and Kay, J H A Simple Electrical Apparatus for the Clinical Treatment of Ventricular Fibrillation, Surgery 30 781 786 1951
5 Hooker D R, Kouwenhoven, W B, and Langworthy, O R The Effects of Alternating Electrical Currents on the Heart Am J Physiol 103 444 454 1933

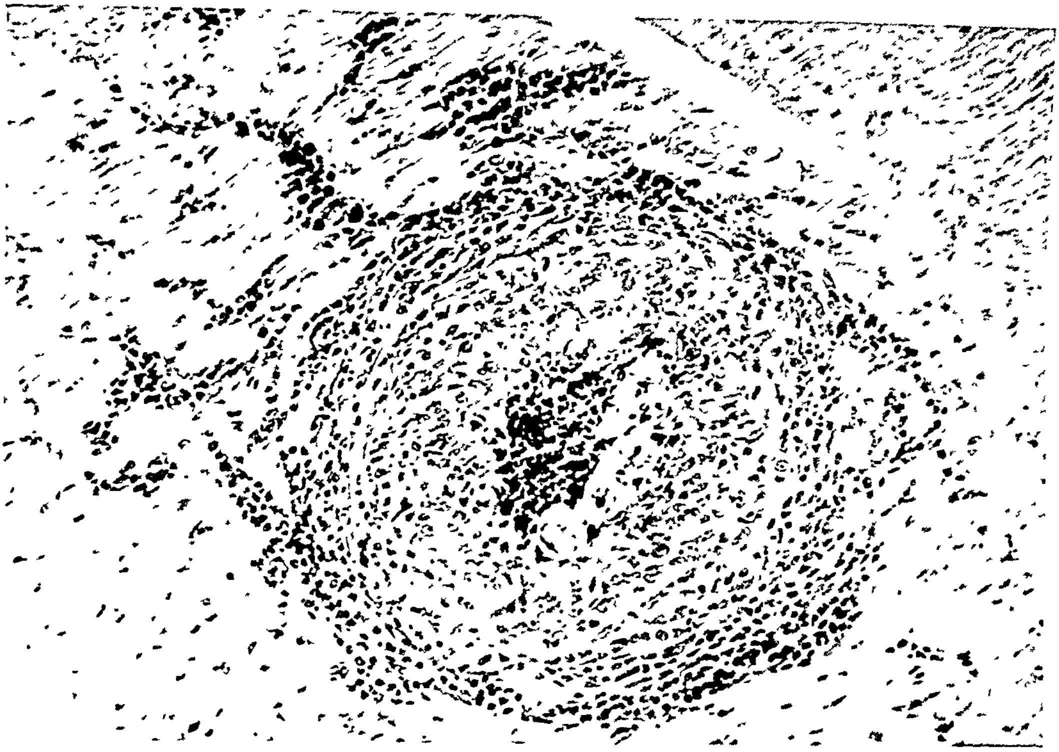


Fig. 1*E*.—Moderate necrosis and inflammation of a coronary artery of the left ventricle twenty-six days postoperatively. The heart was defibrillated with a shock of 230 volts at 1/10 second. (×200.)

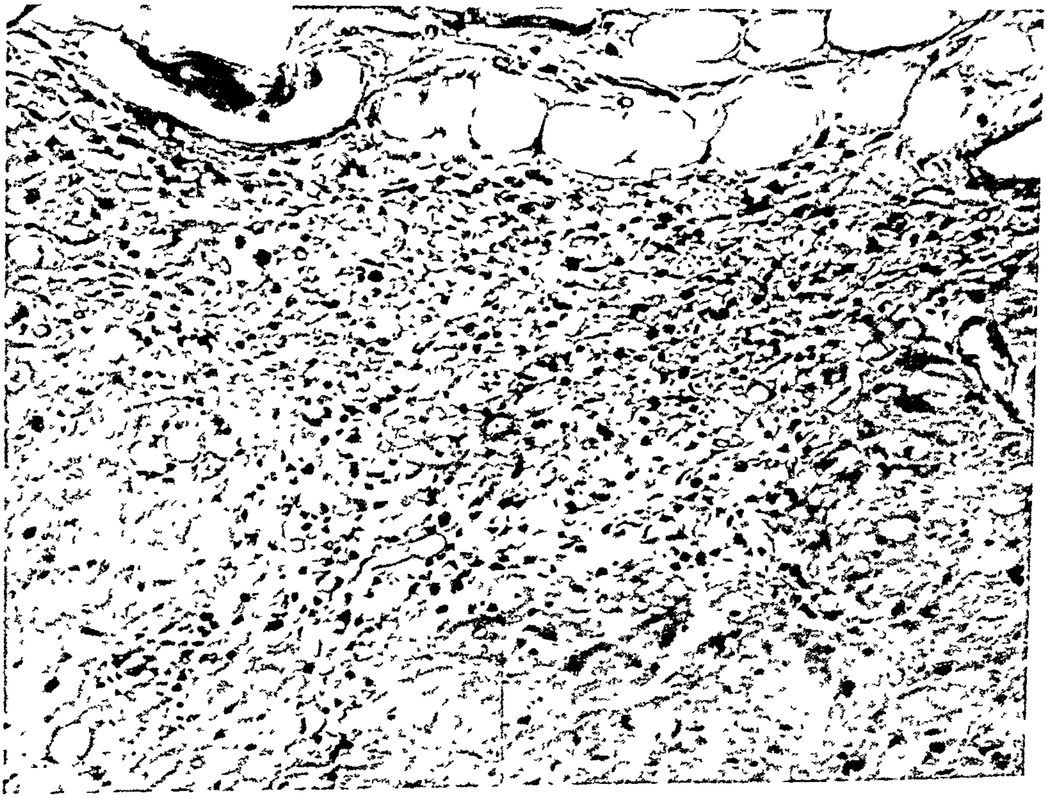


Fig. 1*F*.—Minimal focal subepicardial lysis and degeneration of the myocardium of the left ventricle fourteen days postoperatively. The heart was defibrillated with a shock of 230 volts at 1/4 second. (×200.)

minute correspondence between the heart rate and number of impulses were exact in 90 per cent of the subjects In the other 10 per cent, the same trend of correlation existed but with frequencies above 110 per minute the heart rate failed to follow suit Abrupt change to effective stimulating currents with frequencies above the natural heart rate consistently reproduced the pattern of "take over" by the pacemaker Increase of the pulse width from 10 to 70 milliseconds did not alter the response of the heartbeat but an electrical pulse width of 80 to 120 milliseconds even at low voltages caused pain referable to the chest over the site of the esophageal lead Rhythmic contractions of the upper abdomen due to diaphragmatic stimulation were noted in about one third of the patients These contractions followed the exact rhythm of the stimulator and were eliminated by adjustments on the base plate directing the current

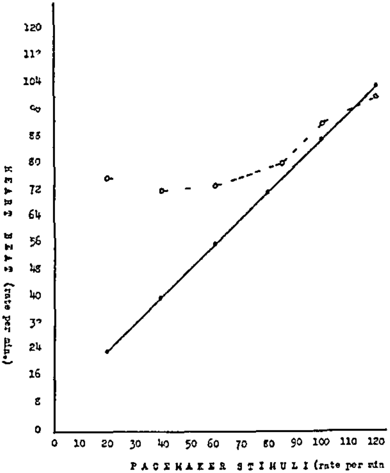


Fig 3—Shows a moderate increase in heartbeat but no correlation with pacemaker stimuli of 20 volts output 20 milliseconds pulse width and frequency of 20 120 impulses per minute

of excitation through other electrodes All the patients tolerated electrical impulses up to the 60 volt level but with higher voltage output pain in the chest was intolerable Ventricular fibrillation due to electrical stimulation did not occur as a complication in this study and harmful clinical effects either immediate or delayed were not noted

*Group II* In all the 10 patients of this group the heartbeat was "taken over" by the pacemaker during the operative procedure for which they were admitted to the hospital The electrical stimulations were repetitive at a frequency of 100 to 120 per minute with a pulse width of 30 [illegible] from a 40 volt output Control of the heart rate comn [illegible]

An experimental test in subjects of Group I consisted of cardiac stimulation by currents derived from output voltages increased in stages from 10 through 100 volts with variations of the pulse frequency and pulse width from the minimum to the maximum setting at each voltage level. In the subjects of Group II, the esophageal lead was introduced just after anesthesia was administered and the proper connections for recording the blood pressure and electrical impulses were made. The pacemaker was set at the beginning of the operation to deliver repetitive stimuli at frequencies greater than the initial heart rate and was interrupted only for brief periods to assess the heartbeat under its own rhythm.

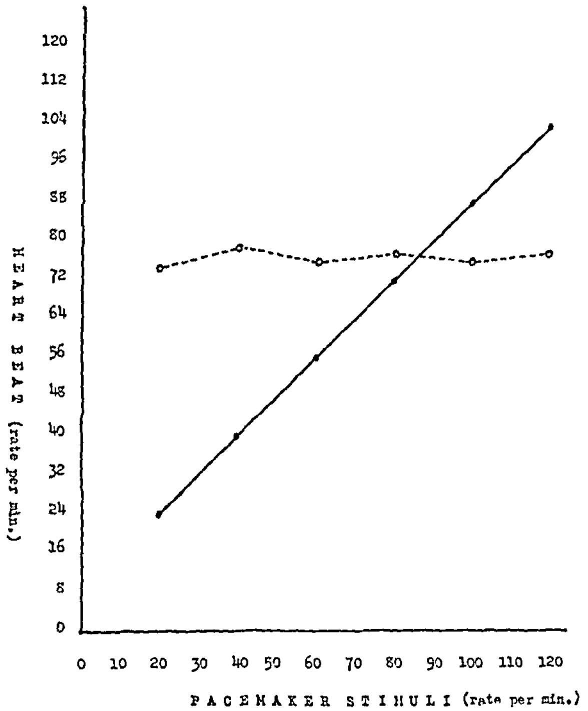


Fig. 2.—Shows no relationship of heart rate to pacemaker stimuli of 10 volts potential, pulse width of 20 milliseconds, and increasing frequency of stimuli from 20 to 120 per minute.

## RESULTS

*Group I:* A total of 54 electrical tests of the heart through all tolerable stimulatory ranges were made in the 16 volunteers. Electrical impulses up to 10 volts with variations up to the maximum in frequency and in pulse width failed to increase the number of heartbeats. Within the 10- to 20-volt range, extrasystoles were occasionally detectable on the pressure pulse tracings. At the 20-volt level a relationship was noticeable between the number of ventricular contractions and the frequency of electrical impulses when such stimulating impulses exceeded 70 per minute. The correlation between heart rate and frequency of stimulation became more nearly equal as the voltage increased above 20 volts. In the 30- to 50-volt range with frequencies of 80 to 120 per administration of anesthesia and external regulation at 100 to 120 beats per minute was continued throughout the entire operative period which averaged 75 minutes. The preanesthetic heart rate averaged 80 per minute and the postoperative rate, after discontinuation of the pacemaker, averaged 94 beats per minute. The operations performed under pacemaker control were limited to appendectomies and herniorrhaphies. Reversion to normal sinus rhythm was immediate in every case. During the period of stimulation the mean blood pressure as denoted by the pressure tracings showed no severe fluctuations or depressions and persisted at a steady level throughout the stimulatory period. Seven patients noted no adverse effects after recovery from the prolonged stimulation under anesthesia and surgery. Three patients complained of a dull ache in the chest which subsided within seventy-two hours.

## DISCUSSION

Electromanometric tracings of the pulse form of the brachial artery established proof of correlation between external stimulation of the heart and ventricular contractions and reflected changes in the peripheral blood pressure during the period of myocardial stimulation. Kymoroentgenograms revealed the increased frequency of auricular and ventricular beats elicited by the pacemaker and showed these to follow in orderly sequence. Electrocardiograms taken at the beginning and end of electrical stimulation of the heart showed no pathologic changes due to prolonged stimulation.

The external pacemaker has been utilized as an effective means of resuscitation of the heart in ventricular standstill but has not been employed previously as a pacemaker to regulate heart action during surgery. On the basis of the present study the latter function is entirely feasible. Complete "take over" of the heart rate throughout an operation can help maintain efficient myocardial contractions and protection against vagal asystole in the poor risk patient. Until a spark-proof electrode is developed, the procedure, as presented here, must be restricted to patients under spinal or intravenous anesthesia. The site for most effective stimulation of the heart has not been definitely ascertained. The sino-auricular node, the auricular-ventricular junction, and the interventricular septum have been proposed, respectively, for optimal stimulation. In patients with normal sinus rhythm, repetitive electrical stimulation in the region of the sino-atrial node would appear to be the most logical site. Except when the conduction system and the cardiac musculature has been damaged organically, as by anoxia, stimulation at this site has generally caused complete myocardial contraction responses and normal pattern electrocardiograms.

Wide variations have been reported in the literature on the values of stimulating electrical currents. For optimum defibrillation of the canine heart, Mackay[4] recommended electrical stimuli of 85 to 170 volts and 1.5 to 3.8 amperes. Zoll[3] used simple monophasic currents of rectangular shape and low voltage for resuscitation of the human heart in standstill [illegible] lopp and Bigelow[2] obtained effective stimulation of the heart [illegible] m 0.01 to 0.1 ampere while Herrod and his associates[5] em [illegible]

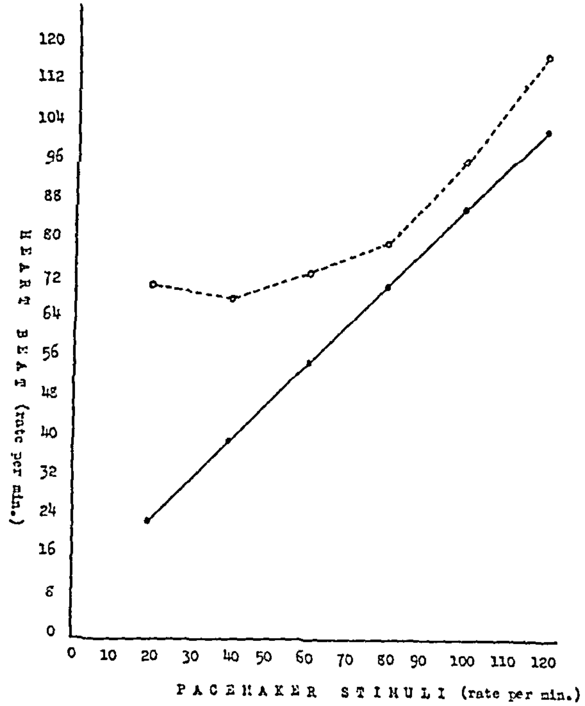


Fig. 4.—Shows correlation of heartbeat and pacemaker stimuli of 40 volts, pulse width 20 milliseconds and frequencies above 80 per minute. Stimuli less than 80 per minute did not affect the heart rate.

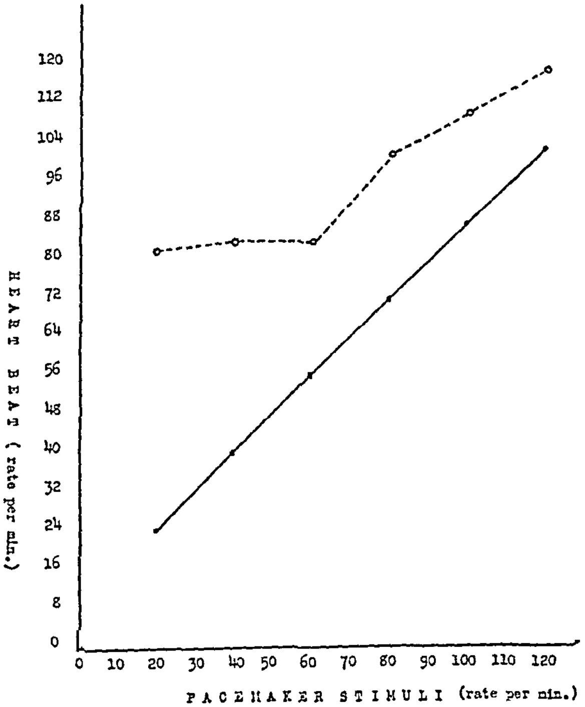


Fig. 5.—Shows almost exact correlation of heartbeat and pacemaker stimuli of 50 volts, pulse width 20 milliseconds and frequencies about 80 per minute. Stimuli delivered below the rate of 80 per minute did not affect the heart rate.

# SOLITARY BENIGN NEUROFIBROMA OF THE LUNG COEXISTENT WITH PULMONARY TUBERCULOSIS

## Report of a Case with Review of the Literature

Daniel W. Dickinson, M.D.,* Wheeling, W. Va., and Harold A. Kipp, M.D.,**
Pittsburgh, Pa.

While intrathoracic neurogenic tumors are fairly common, primary neurogenic tumors, arising and confined within the pulmonary substance, are quite unusual. This report is concerned with a case of benign intrapulmonary neurofibroma associated with active pulmonary tuberculosis.

In 1940, Rubin and Aronson[1] reported finding at autopsy multiple tumor nodules in and on the surfaces of both lungs. These tumors proved to be neurofibromata and no evidence of generalized von Recklinghausen's disease could be found. Bartlett and Adams,[2] in 1946, reported one case of a neurinoma, benign, primary and solitary at the hilum of the left lung. It was firmly attached to the left bronchus, distal to the main bifurcation. A pneumonectomy was performed and an uneventful course followed. There was no evidence of recurrence at the time of writing. Touroff and Sapin,[3] in 1949 reported the surgical removal of a solitary neurofibroma from the apex of the lower lobe of the right lung. It was well encapsulated and readily removed from the lung parenchyma. Diveley and Daniel,[4] in 1951, reported 2 cases of neurogenic tumor, primary in the lung. The first was a neurofibroma, circumscribed, hard and benign, found in the middle lobe of the right lung. The middle lobe was resected and no evidence of recurrence was evident at the time of their report. Their second case was a neurogenic sarcoma in the upper lobe of the left lung, treated by pneumonectomy. This patient died four months after surgery, presumably due to metastases. Lane, Murray, and Fraser,[5] in 1953, described a neurilemmoma of the lung confirmed by tissue culture. This tumor, so termed by A. P. Stout, is thought to arise from the nerve sheath of Schwann and is not to be confused with neurofibroma. However the case described was an example of a solitary, benign, neurogenic tumor arising within the substance of the lung.

## CASE REPORT

S. M., a 33 year old white woman, was admitted to the Pittsburgh Tuberculosis Hospital on Oct. 14, 1954, because of pulmonary tuberculosis detected on routine chest x-ray taken because her brother was undergoing treatment for this condition. The x-ray examination revealed scattering infiltrative disease in the upper portion of the left lung field and haziness

---

From the Pittsburgh Tuberculosis Hospital, Pittsburgh, Pa.

Received for publication April 24, 1956.

*Department of Thoracic Surgery, The Wheeling Clinic, Wheeling, W. Va.

**Chief of Thoracic Surgery, Mercy Hospital, Pittsburgh, Pa.; Associate Professor of Surgery, University of Pittsburgh.

up to 3 amperes. The characteristics (voltage, amperage, etc.) of pacemaker impulses have not been standardized and the variable values noted in the literature can be accounted for by the type of electrical impulse generated, the method of application of the stimulus through the open or closed chest, the resistance of the heart, and other bioelectric effects.

## SUMMARY

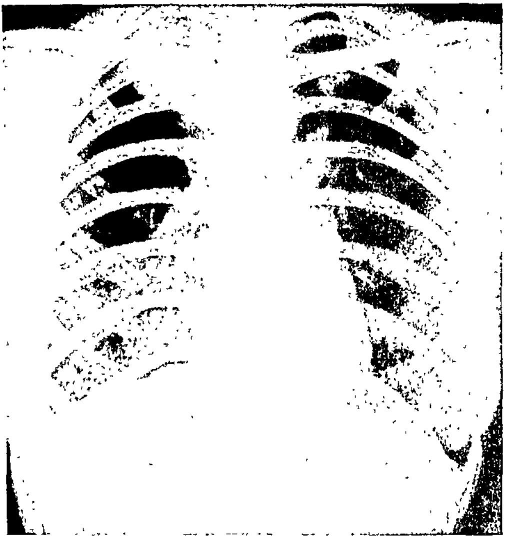
1. Physiologic effects of an electric stimulator on hearts with normal sinus rhythm were studied. Efficient myocardial contractions at a rate greater than the normal sinus rhythm can be produced by means of repetitive electrical stimulation.

2. The heart rate of 16 unanesthetized subjects and 10 anesthetized patients undergoing surgery was controlled externally by means of an electric pacemaker.

## REFERENCES

1. Sweet, W. H.: Stimulation of the Sino-Atrial Node for Cardiac Arrest During Operation, Bull. Am. Coll. Surgeons 32: 234, 1947.
2. Callaghan, J. C., and Bigelow, W. G.: An Electrical Artificial Pacemaker for Standstill of the Heart, Ann. Surg. 134: 8, 1951.
3. Zoll, P. M.: Resuscitation of the Heart in Ventricular Standstill by External Electric Stimulation, New England J. Med. 247: 768, 1952.
4. Mackay, R. S., Mooslin, K. E., and Leeds, S. E.: The Effects of Electric Currents on the Canine Heart With Particular Reference to Ventricular Fibrillation, Ann. Surg. 134: 173, 1951.
5. Hopp, J. A., and Bigelow, W. G.: Electrical Treatment of Cardiac Arrest, Surgery 36: 833, 1954.
6. Herrod, C. E., Lee, R. H., Goggans, W. H., McCombs, R. K., and Gerbode, F.: Control of Heart Action By Repetitive Electrical Stimuli, Ann. Surg. 136: 510, 1952.

Fig. 1.



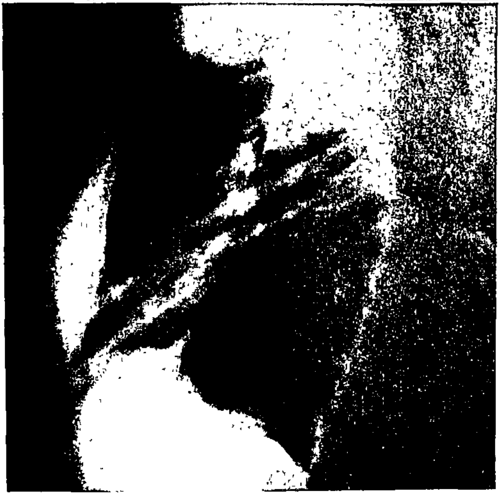


Fig. 2.

Fig. 1.—Posteroanterior projection nine days preoperatively, showing a circumscribe opacity in the apex of the right upper lobe.

Fig. 2.—Lateral projection made at the same time which reveals the posterior postio of the lesion.

high in the apex of the right lung. Blood counts, blood chemistry, blood serology, and urinalysis at regular intervals remained within the normal range. Bronchoscopy had revealed tuberculous endobronchitis of the left upper lobe bronchus. In January, she was operated on for acute appendicitis and recovered without complication. Treatment of the tuberculosis consisted of chemotherapy and routine sanatorium care. The sputum on smear and culture converted to and remained negative by February, 1954, only four months after admission. Bronchoscopy in June, 1954, revealed the left upper lobe endobronchial tuberculosis to be markedly improved. Chest x-rays were taken at appropriate intervals after admission and the lesion in the left lung showed progressive improvement until it appeared quite stable and under control. However, the lesion in the right apex, first described as a haziness and then as a linear density in the outer zone of the second interspace, gradually increased and became circumscribed and was finally described as lemon-sized. Planography on Aug. 13, 1954, showed it to be a solid, noncavitating lesion best delineated well to the posterior (Figs. 1 and 2). At this time, it was felt that this might represent a neoplastic rather than a tuberculous lesion. Bronchoscopic re-examination was performed and the left upper lobe bronchial lesion was found to be well healed and no abnormality of the right upper bronchus was seen. Bronchoscopic washings from the right upper lobe were reported negative for both tubercle bacilli and tumor cells. Other x-ray and fluoroscopic examinations were performed and the consensus of medical and surgical opinions was that surgical exploration was indicated and that the most likely diagnosis was a neurogenic tumor of the chest wall. This was based on the circumscribed appearance of the lesion and its apparent position against the posterior chest wall on the lateral aspect of the vertebral column.

*Operation.*—On Sept. 2, 1954, in the presence of an arrested, contralateral, left upper lobe tuberculous lesion, a right thoracotomy was performed through the bed of the resected fourth rib. A few filmy adhesions over the apex of the upper lobe were divided and the lesion was found to lie in the posterior apical portion of the upper lobe rather than on the chest wall. It was well rounded and approximately the size of a small lemon. It felt soft and almost cystic, similar to a dermoid or inspissated tuberculous lesion, rather than hard or nodular. At its base, near the segmental bronchus, some nodularity was palpable. It was thought that this was a tuberculous lesion and, since it was definitely localized, segmental resection was decided upon. The apical bronchus was isolated and dissected and a partial stenosis was noted a short distance (1 cm.) from its origin, just at the margin of the mass. A second small bronchus, arising immediately medial to that of the apical segmental bronchus, was found to be involved and hence was included in the resection. This probably represented either an anomalous or an accessory apical bronchus, since both the anterior and posterior segmental bronchi were positively identified. The resection proceeded without difficulty and, at the conclusion, the anterior and posterior segments of the upper lobe and the middle and lower lobes expanded readily without evidence of air leak or blood loss.

*Pathologic Report.*—The specimen consisted of the apical segment of the upper lobe of the right lung and a small fragment of bronchial stump. The segment of the lung was collapsed and contained in the apex a rounded, thickened, yellowish nodular area, 2 cm. in diameter. The pleura over this area was grossly thickened but intact. Beneath this white nodular area, there was a small area, 1 cm. in diameter, of necrotic lung tissue containing much blood and fragmented tissue. Also included was a small fragment of bronchial stump which consisted of an ellipse of bronchial tissue curled upon itself. On microscopic examination, sections of the lung revealed a solid neoplastic process located within the parenchyma and partially covered by pleura. The tumor was composed of elongated fusiform fibroblastic cells which in some areas produced small amounts of collagen. The cells were well differentiated and showed no pleomorphism and infrequent mitotic figures. The tumor was moderately vascular and contained irregular thin-walled sinusoidal channels, for the most part, empty of blood. Areas of interstitial edema, interstitial hemorrhage, and early liquefaction were observed. There was no evidence of a tuberculous inflammatory reaction within the adjacent parenchyma. A section of the bronchial stump revealed no abnormalities.

*Diagnosis.*—Neurofibroma of the lung (Figs. 3 and 4).

*Postoperative Course.*—The postoperative course was uneventful. Re-expansion of the remainder of the lung was complete within forty-eight hours and the wound healed per primum (Fig. 5). There was *no evidence of activation of tuberculosis, but she continued* on the antituberculous regime in the hospital until Nov. 6, 1954, because of this possibility. No evidence of recurrence has been found twenty-one months later and the patient has remained well in all respects.

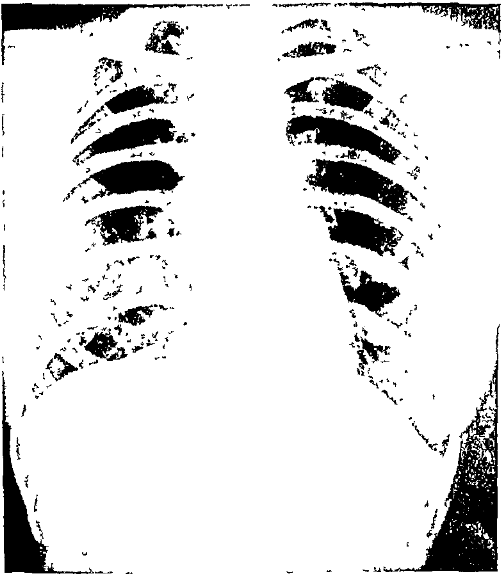


Fig. 5.—A projection made about eight weeks following surgery, which shows complete re-expansion of lung with minimal pleural reaction.

## COMMENT

From the paucity of the literature and the cases reported therein, it is apparent that any neurogenic tumor arising within the lung substance is unusual, and one which is benign and solitary is still more unusual. Of those reported in the literature, one was described as a neurinoma, one as a neurilemmoma, and two as neurofibromata. A third neurofibroma, or the fifth benign neurogenic tumor fulfilling these requirements, is herewith reported. This tumor is notable in view of the fact that it was discovered and seemed to increase in size while the patient was undergoing treatment for active pulmonary tuberculosis in the contralateral lung. It has been rep[illegible]idence

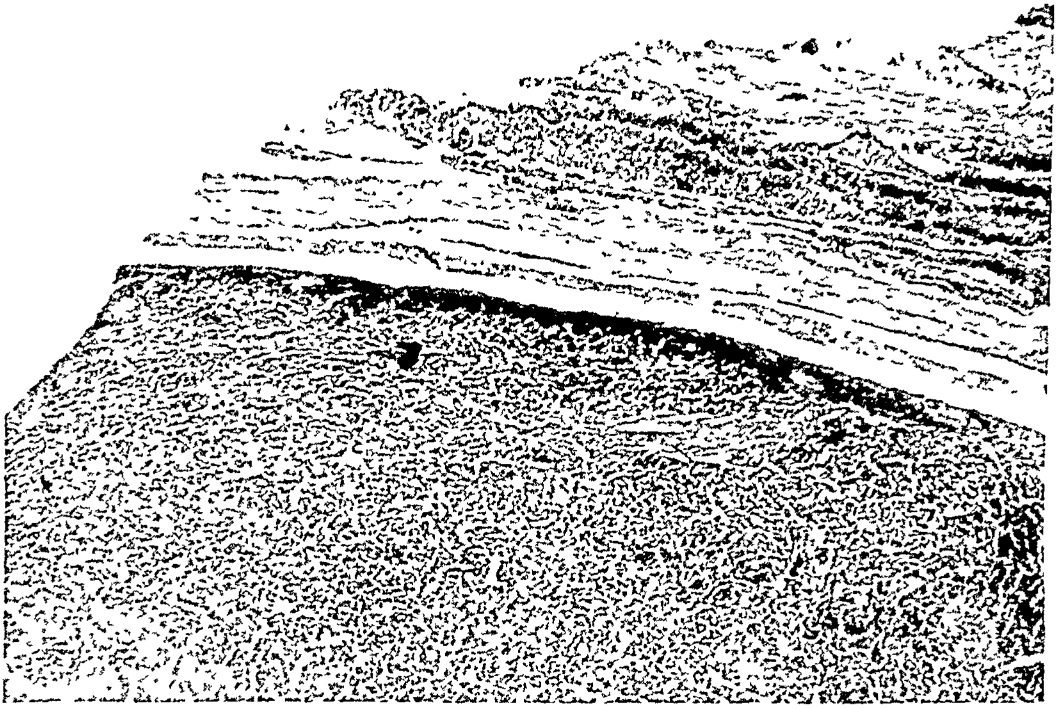


Fig. 3.—The dense cellularity of the tumor occupying the lower half of the illustration is evident. The linear area of dark cells above the capsule of the tumor represents blood within a compressed bronchus (Low-power photomicrograph.)

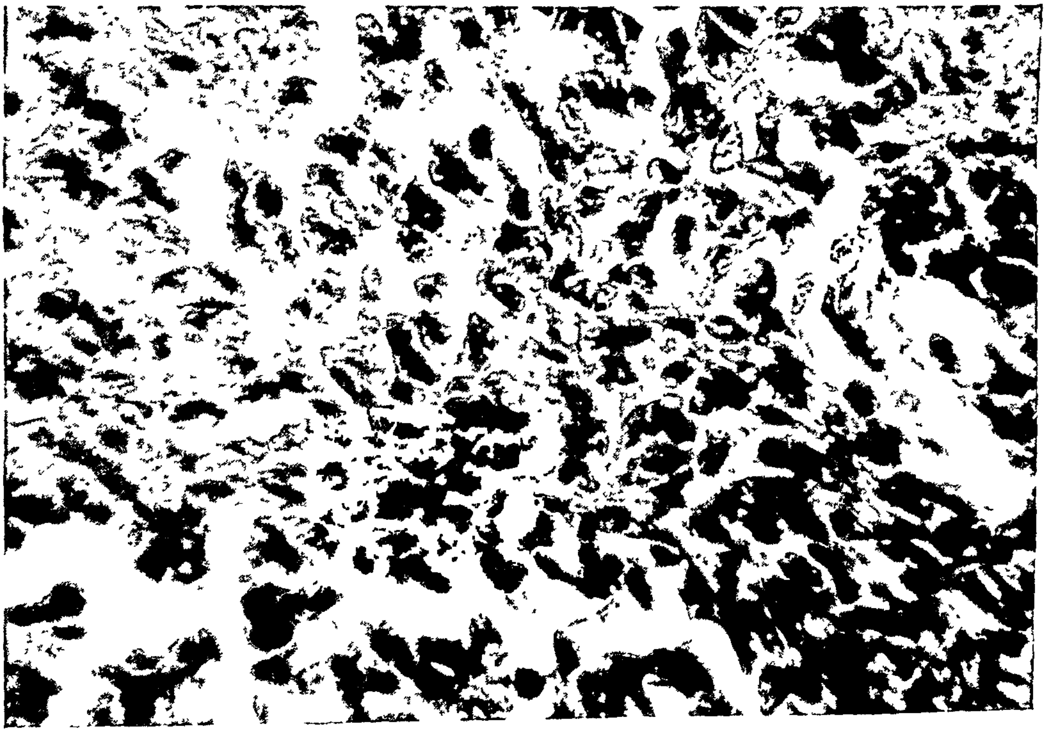


Fig. 4.—Elongated fusiform fibroblastic cells form the entire tumor. (High-power photomicrograph.)

# A BRONCHOSCOPIC TABLE DESIGNED FOR OFFICE USE

Joseph L. Mangiardi, M.D.

Garden City, N. Y.

THE current trend toward outpatient and office bronchoscopy has necessitated many modifications of equipment, depending upon individual taste and facilities. Three years' experience with office endoscopy has influenced me toward a gradual change in concept of a well-functioning bronchoscopy unit with particular emphasis on efficiency and convenience. The necessity for conserving space and personnel has led to progressive concentration of equipment. This has eventuated in the construction of a single unit. This is essentially a converted office examination table, modified, to incorporate the use of a motor-driven headrest, reflector light, power spray, suction system, battery system, and instrument receptacle for bronchoscopic implements. Little or no assistance is needed during the operation and very little space is required. This unit has functioned so smoothly that it appeared worthy of publication.

In Fig. 1, the small size of the table, identical to preconversion size, is demonstrated. It is not conspicuous as a special operating table and can be used for ordinary office examinations. As noted from this view, the headboard has been taken apart, sawed across, and reattached with hinges and reupholstered. The stirrups have been removed. In the left stirrup holder, an adjustable light has been built in for use with the head mirror, when administering anesthesia. Through the right stirrup holder slides a power-spray line, the tip of which prevents it from falling through. When administering anesthesia this is attached to a power-spray bottle. When not in use the line falls back by gravity. It just clears the floor in neutral position.

The patient sits upright on the table during anesthesia. The armboard has been modified by putting a hole in it at an oblique angle. Through this opening a megaphone-like receptacle can be inserted to hold the instruments during bronchoscopy.

In Fig. 2, the headrest and controls are seen as a close-up. The megaphone receptacle is in position. The two paired objects built into the side of the table are battery light controls, each with its own red pilot light to prevent undetected battery wastage when not in use. Adjacent to each pilot light is an off-on switch-rheostat combination. These have a wide range of selectivity, obviating any sudden increase in power which may cause burning out of th bulb. They are situated for convenience within easy reach of the operato

---

From the Division of Thoracic Surgery, Department of Surgery, State University of N. York College of Medicine at New York, and Department of Surgery, N. Y.

Received for publication April 9, 1956.

of bronchogenic carcinoma in autopsied cases of active pulmonary tuberculosis is about 1.4 per cent.[6] However, the association of solitary tumors with pulmonary tuberculosis is quite rare and, since such lesions may well be malignant, surgical exploration and resection to the extent indicated by the nature of the lesion is the treatment of choice. The possibility of associated serious lesions must be kept in mind during the treatment of all tuberculous patients to prevent such lesions from reaching a stage of inoperability before they are recognized.

## SUMMARY

Benign primary intrapulmonary tumors of neurogenic origin are rare within the lung, although similar tumors are found fairly often extrapleurally within the thorax. A case of neurofibroma of the right upper lobe, associated with active pulmonary tuberculosis, is reported. This appears to be the fifth such tumor to be described and the first in which there was an associated pulmonary tuberculosis present. It was of interest that this tumor was seen to expand in size while the patient was undergoing medical treatment for active pulmonary tuberculosis.

## REFERENCES

1. Rubin, E. H., and Aronson, W.: Primary Neurofibroma of Lung, Am. Rev. Tuberc. 41: 801-805, 1940.
2. Bartlett, J. P., and Adams, W. E.: Solitary Primary Neurogenic Tumor of the Lung, J. THORACIC SURG. 15: 251-260, 1946.
3. Touroff, A. S. W., and Sapin, S. O.: Solitary Intrathoracic Neurofibroma, Surgery 26: 787-788, 1949.
4. Diveley, W., and Daniel, R. A., Jr.: Primary Solitary Neurogenic Tumors of the Lung, J. THORACIC SURG. 21: 194-201, 1951.
5. Lane, N., Murray, M. R., and Fraser, G. C.: Neurilemmoma of the Lung Confirmed by Tissue Culture, Cancer 6: 780-785, 1953.

. Nussle, W. F.: Association of Bronchogenic Carcinoma and Active *Pulmonary Tuberculosis*, Dis. Chest 23: 2, 207-216, 1953.

The motor control for the headrest is situated directly beneath the head rest within easy reach of the operator In the horizontal plane the lever is in neutral position, when raised upward the headrest is driven upward, when deflected downward the headrest is lowered The motor can be stopped at any time in either direction by simply returning the lever to the neutral position Headrest position can be varied from 60 degrees in the upward extreme to 90 degrees in the lowered position

It is of additional interest that motor control can be effected by either knee action or finger operation This facilitates head raising or lowering by the operator while looking through the bronchoscope without the sudden or jolting motions often experienced with mechanical systems Another important feature is the utilization of limit switches, applied to the rack which moves the headrest These automatically trip the power when the headrest reaches either extreme position obviating the necessity to manually stop the motor when these danger points are reached

To the right of the motor control are four built-in battery terminals Usually the bronchoscope is powered by the uppermost pair and a telescope from the lower Between the two upper terminals is a blower tip with a removable tight rubber cover when not in operation This was designed for use with the Broyle bronchoscope but I do not use it

Between the lowermost battery terminal is the suction spout which is connected by tubing to the suction tip through a two hole rubber stopper in a specimen jar This is used as a handle to manipulate the suction tip While in the megaphone receptacle, the weight of the jar prevents rotation and sucking up of its contents, although the jar can be emptied at any time by simply turning it upside down When bronchial washings are taken, they are sent to the laboratory in the same jar which is easily detached and replaced by a clean one Likewise, separate washings can be taken from both sides

It should be noted that all controls and operations are within immediate and convenient reach of the operator Incidentally, this table was designed for a right handed surgeon but could be used by a left handed person since the controls are neither delicate nor sensitive

The advantages of the megaphone like receptacle are obvious The suction tip or telescope can be reached for and returned quickly and smoothly without assistance and without having to turn around to reach for them

It is apparent during bronchoscopy that there is no confusion of wires or tubing from table to table or in front of the operator No other pieces of equipment are seen except the table Assistance is usually unnecessary The sum total of these advantages is simplicity which is readily appreciated by the endoscopist It is believed that many of the advantages of the table would still obtain in the operating room

In Fig 3, the interior of the table is seen The cabinet door is open The former cabinet space has been stripped of partitions and former backrest In

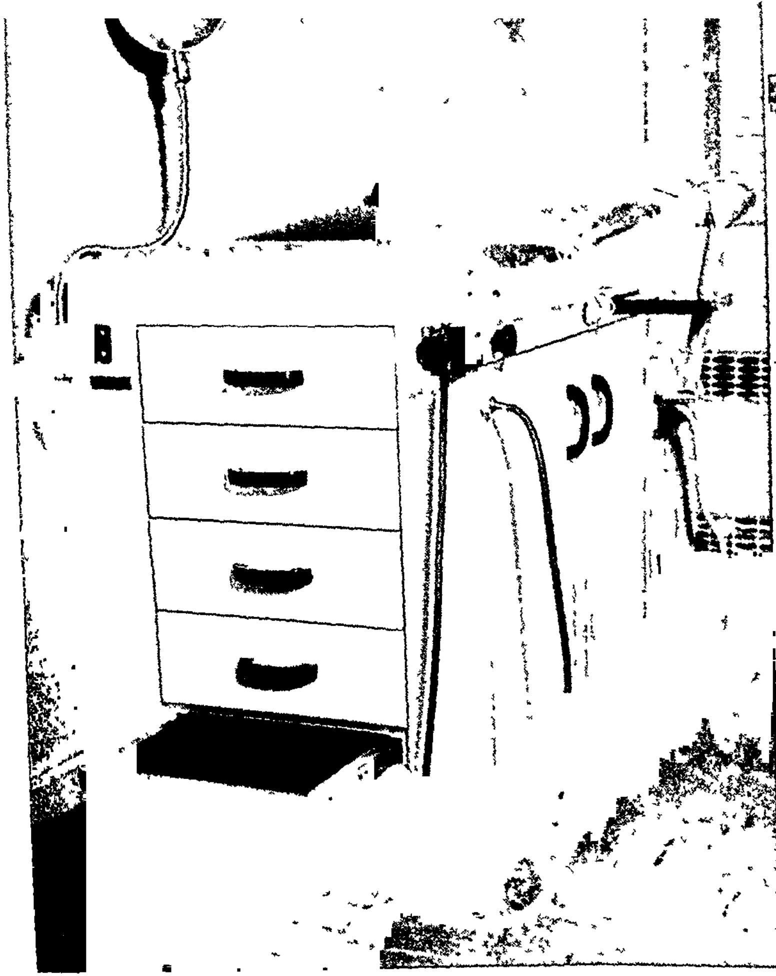


Fig. 1

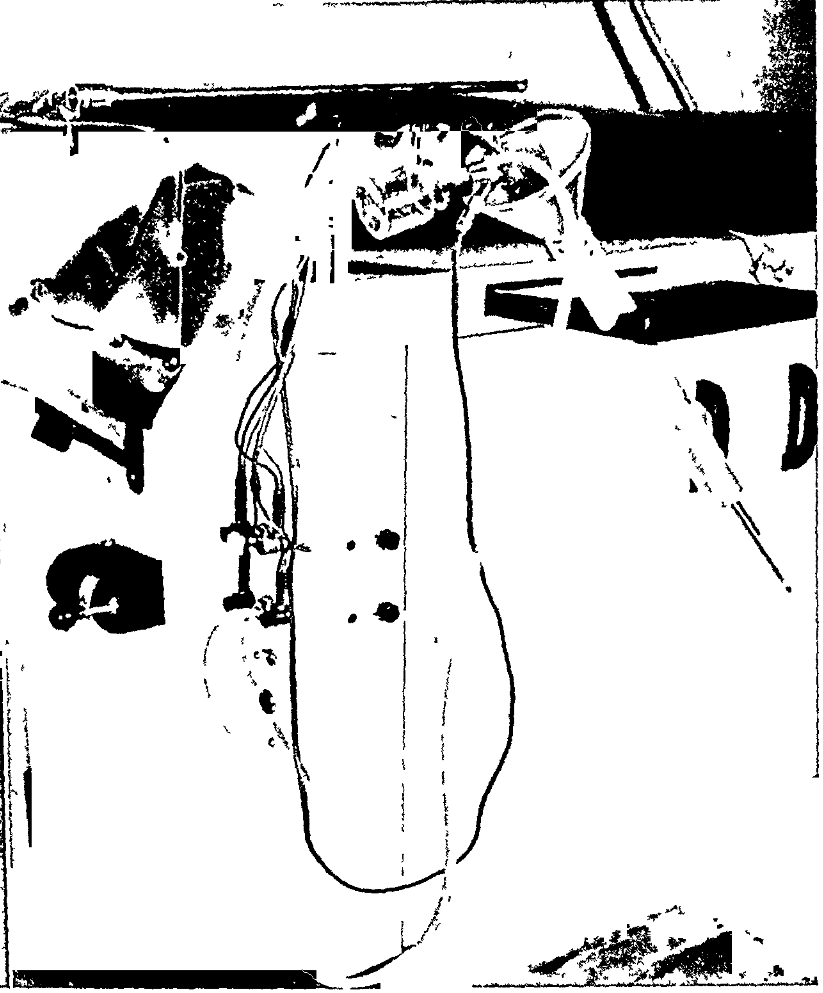


Fig. 2.

Fig. 1.—Rear view of bronchoscopy table Uppermost drawer pulls forward to supply extra table length See text

Fig 2.—Head view of bronchoscopy table This view demonstrates headrest, motor control lever, battery terminals, suction and blower tips, single main switch with pilot light, and twin battery controls with individual pilots Note also megaphone-receptacle in position, containing right-angle telescope and suction tip with built-in collecting jar used as handle

the foreground is a suction blower with appropriate bottles A blower outlet bifurcates, one going to the power spray line at the rear of the table, and one to the control panel

The suction tube extends only to control panel Batteries are on the left and almost hidden from view The suction motor is easily removed To prevent vibrations, the suction motor is heavily padded with sponge rubber In the background is a 1/12 horsepower motor with built in gear box The shaft is supported by a single brace from the bottom of the table On the shaft is mounted a single gear which moves the headrest rack one inch per second, thus, only one external gear is necessary (hidden by suction motor)

Note the elevation limit switch mounted above the rack to trip power at the limit of elevation A similar switch which is hidden by the suction motor, limits depression

In Fig 4, the machine is in operation

### COMMENT

It is not the purpose of this paper to recommend office bronchoscopy in preference to operating room bronchoscopy It may be of interest to state that only a few mild reactions to anesthesia have been encountered Although accidents may occur anywhere avoidance of poor risk patients those with allergic histories, and the use of ½ per cent pontocaine in small amounts has minimized the possibility of reaction An emergency medication—cardiac arrest pack is kept inside the table but fortunately has never been used

Patients are admitted to the office, without breakfast one hour prior to bronchoscopy at which time they are sedated with morphine atropine and Nembutal Following bronchoscopy, they are afforded a rest period of one hour after which they are permitted to leave the office We have found that both the patients and their referring physicians are highly in favor of this as an office procedure

### SUMMARY AND CONCLUSIONS

1 A bronchoscopy table has been designed for office use which has the advantages of motor driven headrest, built in flexible light, built in battery power source, built in suction power spray unit, and a built in megaphone receptacle for instruments

2 Little or no assistance is required during bronchoscopy due to the advantages afforded by the table

3 Complete bronchoscopy can be performed with very small space requirement

4 Office bronchoscopy can be performed with minimal risk and great advantage to both the patient and his physician

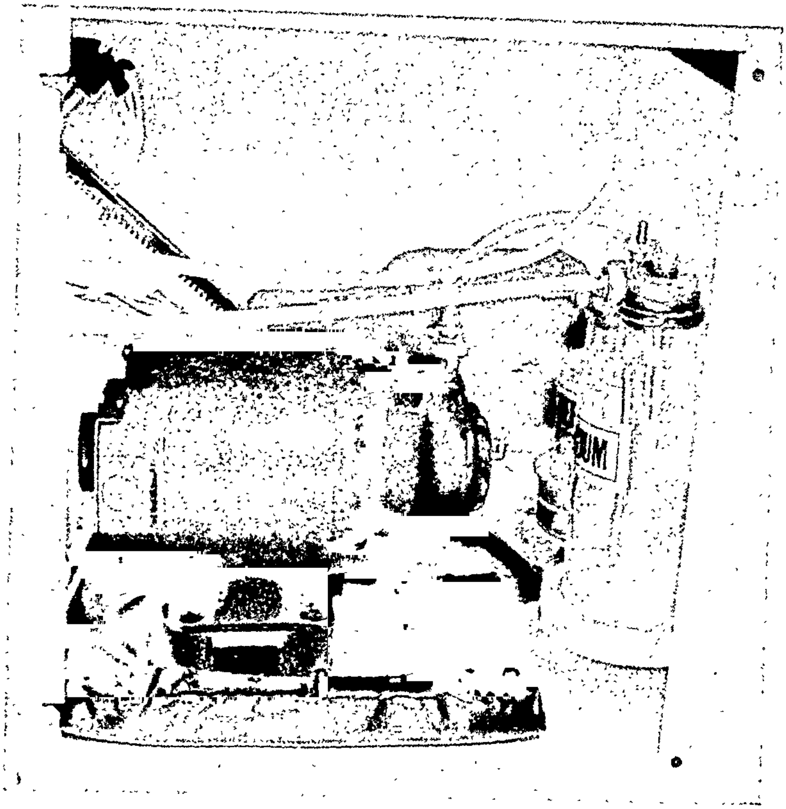


Fig. 3.—Interior of bronchoscopy table. See text.

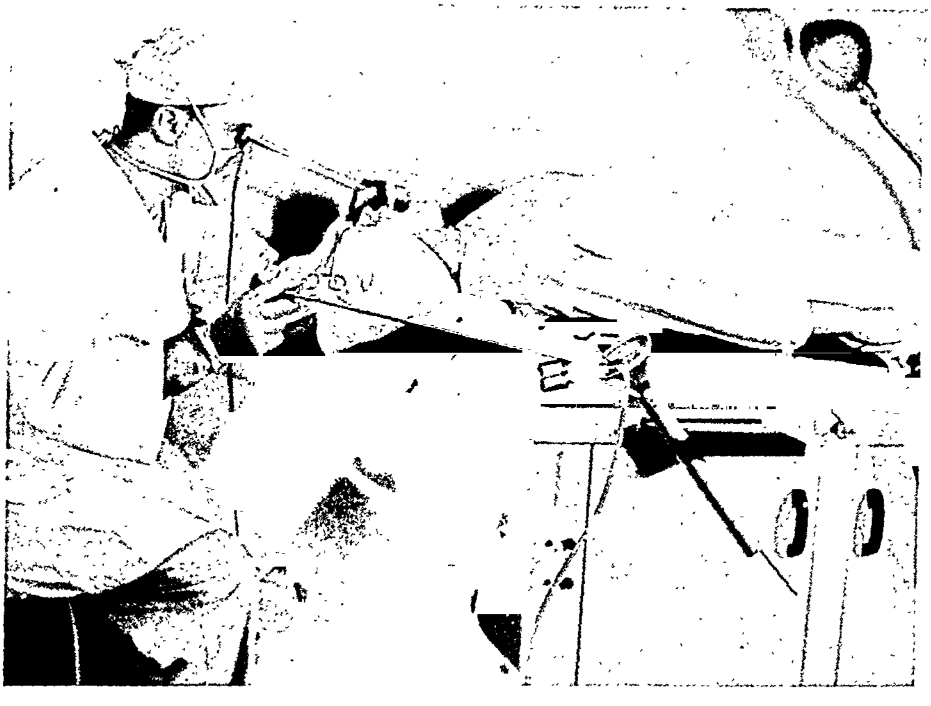


Fig. 4.—Bronchoscopy table in operation. Note controls, megaphone-receptacle with suction tip and built-in sputum jar handle. Note motor control in neutral position. Bronchoscope and right-angle telescope wired and in use.

# THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTY-SEVENTH ANNUAL MEETING
MAY 4, 6, AND 7, 1957
PALMER HOUSE, CHICAGO, ILL.

## Program

### MEMORANDA FOR GUIDANCE OF ESSAYISTS AND DISCUSSANTS

1 In accordance with the By Laws of the Association the papers which are read at the Meeting shall be given to the Reporter *immediately after presentation* If these papers are not submitted promptly their publication may be delayed

2 In publication it is customary to group discussions together on a series of papers [illegible] delay in the return of corrected discussions to the Editor means that all [illegible] papers on the subject will be held up This is manifestly unfair to the [illegible] scientious in the prompt submission of their remarks for publication [illegible] may mean that the discussion will not be published

3 *Regular Program*—Essayists (except in symposia) will be [illegible] to 15 *minutes for* presentation

4 *Thoracic Surgery Forum*—Essayists will be limited to 10 [illegible] for their presentations Papers on the Thoracic Surgery Forum as submitted for [illegible]ation will be limited to 3,000 words At the discretion of the President and [illegible] the limits of the allotted time, one or more discussions per paper will be allowed [illegible]llowing all formal presentations

5 *Discussants*—Discussants will be limited to 3 *minutes* *Only members of the Association* and invited speakers will have the privilege of discussing papers

### MOTION PICTURE SESSION

Motion pictures will be shown on May 4, 5, 6, and 7 Please consult *official program for* titles

*Saturday Morning, May 4, 1957*

8 30 A M Business Meeting

9 00 A M Presentation of Presidential Badge *and Chain of Office by the Society of Thoracic Surgeons of Great Britain and Ireland*

Scientific Session REGULAR PROGRAM

1 **A Roentgen Study of the Evolution of Carcinoma of the Lung.** Le[illegible] G. Rigler, Minneapolis Minn

2 **Intraepithelial Carcinoma in the Tracheobronchial Tree of Lung Cancer Cases** Oscar Auerbach, J. Brewster Gere (by invitation), [illegible]lowski (by invitation), and Arthur Purdy Stout (by invitation), East Orange, N. J.

3 **Bilateral Therapy for Unilateral Spontaneous** *Pneumothorax.* [illegible] Baronofsky, Horace G. Warden (by invitation), [illegible] (by invitation), Joseph [illegible] (by invitation), [illegible] invitation), San Diego, Calif.

4 **The Surgical Treatment of Chronic** *Progressive Pulmonary Emphysema.* John W. Po[illegible] (by invitation), [illegible] (by invitation), [illegible] Mo, and W. V. [illegible], Kansas City, Mo

# Evarts A. Graham

The Editorial Board and Publishers of the JOURNAL OF THORACIC SURGERY announce with deep sorrow the death on March 4 of Dr. Evarts A. Graham, Editor of the JOURNAL since its inception in 1931.

Dr. Graham's guidance was felt in every matter which concerned the JOURNAL. His responsibilities and interests, many of them world-wide, did not preclude his careful reading of manuscripts and his wise and kind direction even in details of editing and publication.

An obituary will be published in an early issue of the JOURNAL.

17. **Alterations in Renal Hemodynamics During and Following Resection of the Thoracic Aorta for Aneurysm.** George C. Morris, Jr. (by invitation), Raymond R. Witt (by invitation), Denton A. Cooley, John M. Moyer (by invitation), and Michael E. De Bakey, Houston, Tex.
18. **Physiological and Pharmacological Studies of Collateral Pulmonary Flow.** David State (by invitation), Peter F. Salisbury (by invitation), and Peter Weil (by invitation), Los Angeles, Calif.
19. **An Experimental Appraisal of the Finney Pyloroplasty in the Prevention of Esophagitis Following the Heller Myotomy.** Paul W. Herron (by invitation), George I. Thomas (by invitation), and K. Alvin Merendino, Seattle, Wash.
20. **Esophageal Motility in Achalasia (Cardiospasm) After Treatment.** Brian Creamer (by invitation), Arthur M. Olsen, Colin B. Holman (by invitation), and Charles F. Code (by invitation), Rochester, Minn.
21. **Postoperative Sodium Excretion Following Administration of Hypertonic Sodium Chloride Solution.** Joseph L. Kovarik (by invitation), and John F. Laws (by invitation). Sponsored by Hiram T. Langston, Chicago, Ill.
22. **Anatomic and Pathologic Studies of the Thoracic Duct.** Harvey W. Kausel (by invitation), Thomas S. Reeve (by invitation), Arthur A. Stein (by invitation), Ralph D. Alley, and Allan Stranahan, Albany, N. Y.
23. **The Study of Ventricular Fibrillation by Threshold Determinations.** Norman E. Shumway (by invitation), John A. Johnson (by invitation), and Richard J. Stish (by invitation), Minneapolis, Minn. Sponsored by F. John Lewis, Chicago, Ill.
24. **Direct Surgical Procedures on the Coronary Arteries: Experimental Studies.** Ormond C. Julian, M. Lopez-Belio (by invitation), and Donald Moorehead (by invitation), Chicago, Ill.
25. **Ligation of the Internal Mammary Arteries as a Means of Increasing Blood Supply to the Myocardium.** Robert P. Glover, and Julio C. Davila (by invitation), Philadelphia, Pa.
26. **The Gradual Closure of Interatrial Defects.** Robert B. Benjamin (by invitation), Robert S. Flom (by invitation), St. Paul, Minn., and F. John Lewis, Chicago, Ill.
27. **Physiological Considerations of Intracardiac Pressures Following Closure of Atrial Septal Defects.** Henry T. Bahnson, and G. Rainey Williams (by invitation), Baltimore, Md.
28. **Transplantation of the Homologous Canine Heart.** Salem F. Sayegh (by invitation), Max Halley (by invitation), and Oscar Creech, Jr., New Orleans, La.

*Monday Afternoon, May 6, 1957*

2:00 P.M. Executive Session. (Limited to Active and Senior Members.)

3:00 P.M. Scientific Session: REGULAR PROGRAM.

Address of the President, Cameron Haight, Ann Arbor, Mich.

**Some Observations on Esophageal Atresias and Fistulas of Congenital Origin.**

29. **Intrapericardial Bronchogenic Cysts: Report of Two Cases and Probable Embryological Explanation.** C. Harwell Dabbs (by invitation), E. Converse Peirce, II (by invitation), Knoxville, Tenn., and Ralph Berg, Jr., Spokane, Wash.
30. **Primary Repair of Traumatic Rupture of the Thoracic Aorta.** Robert G. Pontius (by invitation), Boston, Mass., and Oscar Creech, Jr. New Orleans, La.

5. **A Study of the Role of Drug Resistance in the *Surgical Treatment of Pulmonary* Tuberculosis (Period January 1947—June 1956).** William R. Sweetman (by invitation) and John M. Salyer, Denver, Colo.
6. **The Value of Routine Pulmonary Function Studies in Thoracic Surgical Cases.** Hurst B. Hatch, Jr. (by invitation), Joseph K. Bradford (by invitation), and Alton Ochsner, New Orleans, La.

*Saturday Afternoon, May 4, 1957*

2:00 P.M. Scientific Session: REGULAR PROGRAM.

7. **Rigid Plastic Prostheses in Vascular Surgery.** F. X. Byron, Josh Fields (by invitation), Augustus Foster (by invitation), and Richard Hood (by invitation), Beverly Hills, Calif.
8. **Diagnostic and Physiologic Measurements Using Left Heart Catheterization.** W. S. Blakemore (by invitation), T. G. Schnabel (by invitation), P. T. Kuo (by invitation), and H. B. Conn (by invitation), Philadelphia, Pa.
9. **Effect of Exercise on the Diastolic Atrio-Ventricular Gradient in Mitral Stenosis.** Robert S. Litwak (by invitation), Philip Samet (by invitation), W. H. Bernstein (by invitation), Leonard Silverman (by invitation), Hyman Turkewitz (by invitation), and Milton E. Lesser (by invitation), Miami, Fla.
10. **Surgical Treatment *of Transposition of the Aorta and Pulmonary Artery.*** Thomas G. Baffes (by invitation), William L. Riker (by invitation), Arthur DeBoer (by invitation), and Willis J. Potts, Chicago, Ill.
11. **The Use of a Mechanical By-Pass During Cross-Clamping of the Aorta.** Harold King (by invitation), and Harris B. Shumacker, Jr., Indianapolis, Ind.
12. **Elective Cardiac Arrest: An Adjunct to Open Heart Surgery.** Donald B. Effler, Laurence K. Groves (by invitation), Harold F. Knight (by invitation), Wilhelm J. Kolff (by invitation), and F. Mason Sones, Jr. (by invitation), Cleveland, Ohio.
13. **Experiences With the Use of Cardioplegia (Induced Cardiac Arrest) in the Repair of Interventricular Septal Defects.** Conrad R. Lam, Thomas Geoghegan (by invitation), Charles Sergeant (by invitation), and Edward Green (by invitation), Detroit, Mich.

6:30 P.M.—8:30 P.M. COCKTAIL PARTY. Informal. Palmer House.

*Sunday, May 5, 1957*

No Scientific Session

For Motion Picture Session consult official program.

*Monday Morning, May 6, 1957*

8:30 A.M. Scientific Session: *THORACIC SURGERY FORUM.*

14. **The Effects Produced by Various Types of Pump Oxygenators During Two-Hour Partial Perfusions in Dogs.** Marian E. Molthan (by invitation), Stanley Gianelli (by invitation), Richard J. Best (by invitation), James A. Dull (by invitation), and Charles K. Kirby, Philadelphia, Pa.
15. **Acid-Base Balance During Prolonged Cardio-Respiratory By-Pass.** Matthias Paneth, Traveling Fellow of the Association, 1956-57 (by invitation), London, England, M. Nazih Zuhdi (by invitation), and William Weirich (by invitation), Minneapolis, Minn.
16. **Coronary Perfusion as an Aid to Open Heart Surgery Under Hypothermia in Man.** J. V. Maloney, Jr. (by invitation), S. A. Marable (by invitation), and W. P. Longmire, Jr., Los Angeles, Calif.

The Journal of
Thoracic Surgery

Vol. 33 May, 1957 No. 5

# Original Communications

---

## A REVIEW OF 34 CONSECUTIVE CASES OF PNEUMONECTOMY, WITH AND WITHOUT CONCOMITANT THORACOPLASTY, IN THE TREATMENT OF TUBERCULOSIS

John L. Shek, M.D.,* and Gordon D. Myers, M.D.**
Rome, Ga.

GREAT strides have been made in the surgical treatment of pulmonary tuberculosis since the concept of resection was brought into the recent literature by Freedlander,[1] in 1935. Jones and Dolley,[2] Janes,[3] and Sweet[4] followed up with reports of lobectomy and pneumonectomy in the treatment of tuberculosis in the ensuing ten years. Overholt[5] and Chamberlain[6] and their associates, applied the knowledge of segmental anatomy of the lung in the resection for the disease, as Churchill and Belsey,[7] and Blades[8] had first made use of it in excisional surgery for bronchiectasis. With the improved knowledge of anesthesia, blood transfusion, pulmonary physiology and anatomy, thoracic surgery, the introduction of antituberculous drugs, and a better concept of tuberculous pathology, pulmonary resection of different magnitude has well established itself as another definitive surgical treatment for tuberculosis. Attention, therefore, has been focused more toward the refinement of surgical technique and postoperative care to improve further the already noteworthy results.

Literature has been abundant with reports of series of cases of resection for the treatment of pulmonary tuberculosis. It has become apparent that the most dreaded complication and the major cause of postoperative, and even delayed mortality, is the notorious bronchopleural fistula. Although faulty technique in the closure of the bronchus stump could, and does, occasionally account for the fistula formation, it is, however, agreed that the intrinsic disease

---

Received for publication April 2, 1956.

*Chief of Surgery, Battey State Hospital, Rome, Ga.

**Resident in Thoracic Surgery, Battey State Hospital, Rome, Ga.

31. **The Electro-encephalogram in Patients Undergoing Open Intracardiac Surgery With the Aid of Extracorporeal Circulation.** Richard A. Theye (by invitation), Robert T. Patrick (by invitation), and John W. Kirklin, Rochester, Minn.

*7:00 P.M.* *COCKTAILS, BANQUET, AND DANCING,* Palmer House.

Attendance Limited to Members of the *Association and their ladies*, invited speakers and their ladies.
Dinner dress preferred.

*Tuesday Morning, May 7, 1957*

9:00 A.M. Scientific Session: REGULAR PROGRAM.

32. **Cine-Fluorography in the Evaluation of Neuromuscular Diseases of the Esophagus.** *Frederick S. Cross, Earle B. Kay, and George F. Johnson (by invitation),* Cleveland, Ohio.
33. **Esophageal Hiatus Hernia of the Diaphragm: An Analysis of Surgical Results.** *George H. Humphreys II, Jose M. Ferrer, Jr. (by invitation), and* Philip D. Wiedel (by invitation), New York, N. Y.
34. **An Operation for Hiatus Hernia With Short Esophagus.** J. Leigh Collis (by invitation), Birmingham, England.
35. **Traumatic Rupture of the Diaphragm—Clinical Manifestations and Surgical Treatment.** Gerard Desforges (by invitation), John W. Strieder, Joseph P. Lynch, and Irving M. Madoff, Boston, Mass.
36. **Reconstruction of the Esophagus With Segments of the Colon.** William E. Neville (by invitation), and George H. A. Clowes, Jr., Cleveland, Ohio.
37. **Primary Tumors of the Heart: A Surgical Problem.** J. Gordon Scannell, and Hermes C. Grillo (by invitation), Boston, Mass.

*Tuesday Afternoon, May 7, 1957*

*[illegible]:00 P.M.* *Scientific Session: REGULAR PROGRAM.*

38. **Ventriculoplasty for Cardiac Aneurysm.** C. P. Bailey, H. E. Bolton (by invitation), and R. A. Gilman (by invitation), Philadelphia, Pa.
39. **Visual Repair of Congenital Aortic Stenosis During Hypothermia.** Henry Swan, S. Gilbert Blount, Jr. (by invitation), and Robert H. Wilkinson (by invitation), Denver, Colo.
40. **Surgical Treatment of Stenotic or Regurgitant Lesions of the Mitral and Aortic Valves by Open Cardiotomy.** C. Walton Lillehei, Richard A. DeWall (by invitation), Vincent L. Gott (by invitation), and Richard L. Varco, Minneapolis, Minn.
41. **The Surgical Correction of Aortic Insufficiency.** Warren J. Taylor (by invitation), Wendell B. Thrower (by invitation), Harrison Black, and Dwight E. Harken, Boston, Mass.
42. **The Clinical and Physiological Criteria for the Surgical Correction of Mitral Regurgitation.** Julio C. Davila (by invitation), P. Jumbala (by invitation), Robert G. Trout (by invitation), and Robert P. Glover, Philadelphia, Pa.
43. **The Application of Hydraulic Principles to the Coronary Circulation.** Bernard L. Brofman (by invitation), and Claude S. Beck, Cleveland, Ohio.

through the usual posterolateral incision and the pleural cavity is entered through fifth rib bed Great caution is exercised in avoiding rupture into the tuberculous cavity and extrapleural dissection is used extensively, if necessary, without concern about maintaining an intact parietal pleura Oozing from the raw surface of the chest wall is usually stopped by warm saline packs and this often ceased after removal of the ribs The lung is resected in the usual fashion At this time, the general condition of the patient is estimated and the blood loss calculated by the sponge weighing technique and the operative time is surveyed If the patient's condition is satisfactory, local intercostal nerve block with one per cent procaine was carried out prior to subperiosteal resection of the second down through the seventh ribs Posteriorly, the resection starts from within the lateral margin of the spinalis group of muscles, at times from the level of the transverse processes, anteriorly, to the midclavicular line of the second rib, in diminishing lengths to the seventh rib, which is around the scapular line so as to allow the shoulder blade to fall in without any impingement The usually shredded intercostal muscle bundles are then repaired to re form a more or less complete partition between the pleural space and the subscapular space A certain amount of tailoring is done to these bundles to reduce the size of the residual pleural space One intercostal drainage tube is left inside the space connected to a underwater seal system A tight dressing with a roll of cotton padding over the decostalized portion of the chest wall under the axilla is then applied to further immobilize the side and reduce the amount of possible mediastinal fluttering Adequate tracheobronchial aspiration is then done, usually without the necessity of bronchoscopy

There is no special feature in the postoperative care Primary attention is paid to the amount of drainage, which is measured, and the blood loss is adequately replaced The tube is left in for 24 to 48 hours and longer in cases where contamination is suspected The pressure dressing sometimes is necessary for about two weeks, postoperatively

This is a series of 34 pneumonectomies done consecutively between May 1953, until February, 1955, during which time a total of 225 resections of various extent were done, a percentage of 15 1 These patients, therefore all have had a postoperative follow up of at least 9 months We are quite aware that due to the miraculous effect of antituberculous drugs in a majority of cases, total resection of lung has become less and less indicated With the improvement of surgical technique, most of the cases that would have called for a pneumonectomy in the earlier days of surgical excision now require only lobar, or lobar and segmental resection However, all the patients in this series had a totally destroyed lung without a single segment that was worth salvaging They all had had bilateral involvement at one stage The less involved side not infrequently had had cavitary disease, but those cases had improved and stabilized, while the side with the major involvement progressed on to a stage of total destruction in a few cases either in the form of residual giant air containing cystic remnant of lung, or, more often, to a unilocular or multilocular cavitary residue with patches of pneumonic changes Most of these cases were considered to be only salvageable Table I will show the distribution of cases in the two groups, one with concomitant thoracoplasty and the other with nonconcomitant thoracoplasty In the nonconcomitant group, there were 2 thoracoplasty failures, one plombage failure, one primary pneumonectomy with postresection thoracoplasty, and 13 cases with preresection thoracoplasty (The advantages of preresection thoracoplasty have been elaborated upon in another communication )[23] in the bronchial wall and infection in the pleural space surrounding the amputated stump are responsible for its incidence in most cases. It has also become increasingly clear that failure to properly cope with the residual pleural space following resection of lung tissue usually precedes this complication. Thoracic surgeons working in this field have employed various means to handle the residual space, but no single solution has been agreed upon as yet. Many have explored the use of foreign body prosthesis, intrapleurally, extrapleurally, or extramusculoperiosteally, such as oil, Sirconium, Lucite sphere, Gelfoam, Ivalon sponge, cellulose, Neoprene, Polyvinyl sponge, etc.[9-17] Some used air in the form of pneumothorax and pneumoperitoneum. These either have a limited usefulness, the acceptance has been controversial, or they have been proved to be undesirable. The only other recourse left to cope with the residual pleural space for the sake of lowering the likelihood of bronchopleural fistula and prevention of overdistention of the remaining lung tissue, ipsilateral and/or contralateral, lies in the versatile thoracoplasty. There are many varied opinions regarding the desirability of thoracoplasty in conjunction with pulmonary resection. This paper is concerned only with the concomitant thoracoplasty and pneumonectomy.

Graham, in 1933, performed the first successful pneumonectomy and concomitant thoracoplasty. Iverson and Skinner[18, 19] reported its use in pulmonary tuberculosis. Samson,[20] Conklin,[21] and Cruickshank[22] and their associates followed with reports of encouraging results in selected cases. The advantages of the one-stage operation are well elaborated upon by these authors in their original articles. However, because of the greatly increased morbidity, such combined procedure has not gained general acceptance and its usefulness is, therefore, limited to strictly selective cases. However, we believe that this procedure is stigmatized without being duly investigated.

This paper reports a comparative study of 17 cases of pneumonectomy and concomitant thoracoplasty and another 17 cases of pneumonectomy without concomitant thoracoplasty performed consecutively over a period of 21 months, ending February, 1955. From a review of the various data, the authors are not particularly impressed with the increased morbidity in the postoperative period. In fact, it is felt that the postoperative course is comparatively more benign than in operations for primary pneumonectomy done without concomitant thoracoplasty. An attempt is made first to establish the fact as to whether the two groups are of comparable composition regarding the factors of race, age, sex, extent of disease, activity of lesion, and length of preoperative antibiotic treatment, etc. The two groups are then compared as to their operative and postoperative morbidity. Various favorable features of the combined procedure are also noted.

## TECHNIQUE

The *face-down* position is used. The anesthesia consists of Pentothal sodium and Anectine induction with endotracheal intubation as soon as possible, and supplemented with nitrous oxide, oxygen mixture, and intravenous drip Anectine. Exposure is gained cavitation, granulating tissue, endobronchial disease, or even caseous pneumonic process. The expression inactive disease is used to designate lack of such findings, as well as cases where only dormant fibrocaseous residue was present. Instances where only the controversial "healed cavity" or giant air-containing spaces of the lung were found are also regarded as inactive disease.

TABLE II

| | PATHOLOGIC PROCESS | | BACTERIOLOGY OF RESECTED SPECIMEN | | | | | |
|---|---|---|---|---|---|---|---|---|
| | ACTIVE DISEASE | INACTIVE DISEASE | D. S. + | CUL. + | D. S. + | CUL. − | D. S. − | CUL. + | D. S. − | CUL. − |

| | ACTIVE DISEASE | INACTIVE DISEASE | D. S. + CUL. + | D. S. + CUL. − | D. S. − CUL. + | D. S. − CUL. − |
|---|---|---|---|---|---|---|
| Combined procedure | 13 | 4 | 7 | 5 | 1 | 4 |
| Separate procedure | 15 | 2 | 8 | 6 | 1 | 2 |

D. S. = direct smear.
CUL = culture.

This was especially considered to be so when bacteriologic studies failed to show presence of tubercle bacilli. No special technique was employed to cultivate the bacteria, although some of them were incubated for a longer period of time than the routine six weeks. Again, the data in Table II does not show any striking difference in results in the 2 groups.

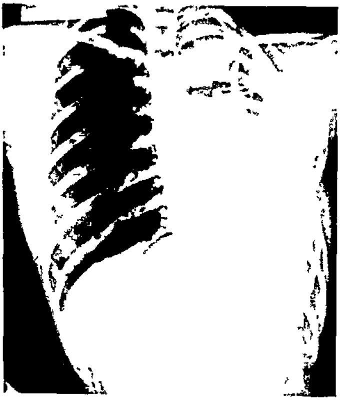


*Fig. 3*—Case No. 27, R. L. S., a 28-year-old white woman, was admitted Sept. 28, 1945, far advanced, D. She received numerous course [illegible] f streptomycin, INAH, and PAS, preoperatively. Left anterior and [illegible] was performed on Aug. 20, 1948; [illegible] 26, 1955 [illegible] cor pulmonale for two weeks [illegible] broke [illegible] onths. Sputum was positive on J [illegible] orted since. Specimen shows destroyed lung with large lower lobe [illegible] active endobronchitis. The patient is now in process of being

In respect to preoperative antituberculous drug therapy, all 34 patients have received treatment in various combinations over a period of time, averaging 26.1 months, with 2 cases of six months' duration and 3 cases over three years. A minimum of eighteen months of drug therapy followed the resection. Bacteriologically, drug resistance assay was done in 23 of the 34 cases and

TABLE I

|  | NUMBER OF CASES | SEX |  | RACE |  | AGE GROUP |  |  |  | AVERAGE LENGTH OF DISEASE YEARS |
|---|---|---|---|---|---|---|---|---|---|---|
|  |  | M | F | WHITE | NEGRO | 16-19 | 20-29 | 30-39 | 40-49 |  |
| Combined procedure | 17 | 2 | 15 | 5 | 12 | 4 | 8 | 4 | 1 | 3.7 |
| Separate procedure | 17 | 5 | 12 | 5 | 12 | 0 | 7 | 6 | 4 | 4.4 |

From Table I, it became at once apparent that the two groups are closely comparable as to the distribution of sex, race, and the average duration of disease, which was measured from the date when the diagnosis of pulmonary tuberculosis was established until the date of definitive operation. It indicates that, although the majority of patients in either group lies within the third and fourth decade of life, there was a tendency of the group with the combined procedure to be in the younger age limit.

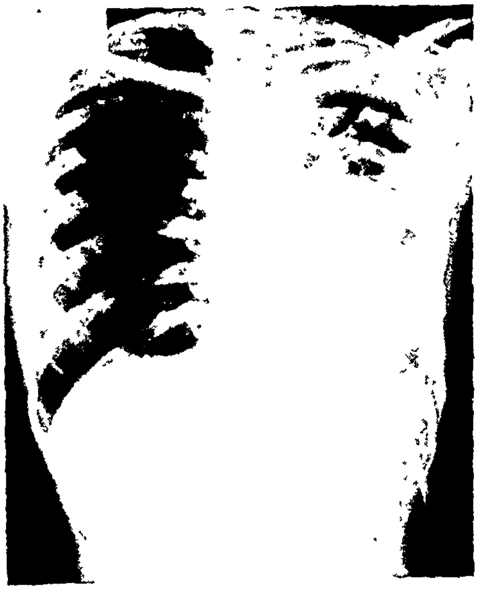


Fig. 1.

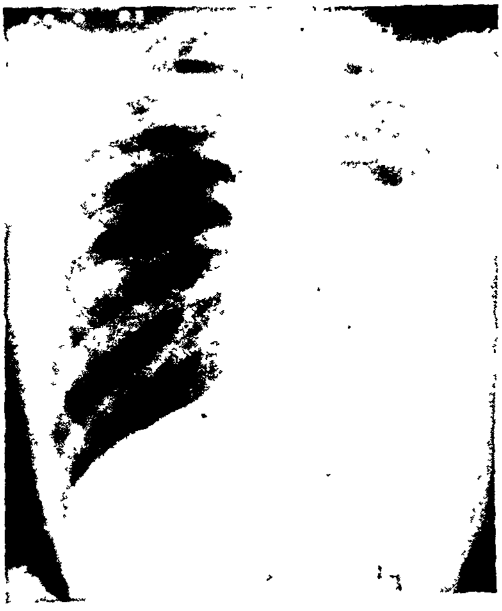


Fig. 2.

Fig. 1.—Case No. 22. P. W., a 37-year-old Negro woman, was admitted Nov. 10, 1950, far advanced, D. She received 14 months of streptomycin and para-amino-salicylic acid (PAS), and 14 months of isonicotinic acid hydrazide (INAH) and PAS, preoperatively. Combined pneumonectomy and thoracoplasty were performed on Feb. 2, 1954, with uneventful recovery. The sputum positive, Jan. 22, 1954, and converted immediately and has remained so since. The specimen shows destroyed lung with three large cavities, active endobronchitis, and caseous bronchopneumonia. The patient was discharged home, Dec. 28, 1954.

Fig. 2.—Case No. 11. W. H. W., a 26-year-old Negro man, was admitted Sept. 8, 1952, far advanced, C. He received 12 months of streptomycin and PAS and six months of INAH and PAS, preoperatively. A combined procedure was performed on Oct. 21, 1954, with smooth recovery. The sputum was positive last on Feb. 18, 1954, and has remained negative since. Specimen shows healed cavity with destroyed lung and no active tuberculosis. He was discharged April 26, 1955.

The following table (Table II) compares the degree and variety of pathologic processes from the resected specimens in both groups. It shows that the two groups also have more or less similar data. In Table II, by active disease we mean that there is definite active tuberculous process in the form of either

The findings so far tabulated indicate that the two groups of patients who have had a pneumonectomy can be closely compared with regard to sex, race, age group, extent of disease activity of disease, length of illness prior to surgery, and length of preoperative and postoperative chemotherapy It would, therefore, seem worth while to make a comparison of the operative and postoperative morbidity in the two groups in an effort to uncover some indication as to whether the concomitant procedure is as risky an operation as it is reputed to be

From the data in Table IV, which listed postoperative complications, there is no predominance of morbidity rate in either group The 2 cases of prolonged low grade fever, lasting for about one month postoperatively are believed to be due to a tuberculous process in the mediastinal lymph nodes which subsides under treatment with antibiotics without consequence The one bronchopleural fistula, which developed in the second week, postoperatively was treated with revision thoracoplasty, including resection of transverse processes in the third week The fistula apparently closed in the fourth week but the subscapular infection persisted, leaving behind draining sinuses along the incision at the present time The sputum, however, converted six months, postoperatively, and has remained so since then which is about five months One patient developed a localized tuberculous abscess along the incision and healed after incision and drainage and topical application of isonicotinic acid hydrazide (INAH) solution All 4 patients who developed transient episodes of right heart failure had extensive bilateral involvement and one of them had miliary tuberculosis and proved meningitis They all responded readily to digitalization and other supportive measures and recovered within one to two weeks One, who had had both anterior and posterior Monaldi drainage done seven years before, had the anterior wound break down after the resection It healed after five months of local treatment All patients had some degree of hyperpnea, and a few had dyspnea in the first 48 hours, but none were cyanotic for more than a few minutes The tuberculous pericarditis occurred in the fourth month postoperatively, in the patient who eventually died in the fifth month The data of these late postoperative complications, therefore compare favorably between each group Upon reviewing the literature, it is noticed that they compare quite well with that of O'Brien and his co workers[24] who reported a series of 49 pneumonectomies for tuberculosis done between 1947 and 1952, with a morbidity rate of both early and late complications of 43 per cent and five deaths McBurney and his associates[25] reported 20 cases of pneumonectomy for tuberculosis performed through December, 1952, with complications in 13 patients, 3 had bronchopleural fistulas, and there were three deaths Baffes and associates,[26] reviewed 72 pneumonectomies done between 1942 and 1951, and found 14 deaths, 3 cases of empyema, and 7 of spread Although it is realized that these reports concern operations performed in the earlier days of pulmonary resection for tuberculosis, it is felt that in spite of the limited size of this series done between May, 1953, and February, 1955, that the postoperative morbidity does not compare unfavorably with pneumonectomy done without concomitant thoracoplasty showed the usual pattern of conversion of organisms into resistant strains. Nineteen patients had preoperative conversion of sputum, with an average length of about 14.2 months before surgery. The sputum of 12 patients was converted directly and immediately after surgery, and in 2 converted six and seven months, respectively, subsequent to resection. Only in one case (2.9 per cent) has it remained persistently positive to culture, apparently from an ulcerated bronchial stump, diagnosed bronchoscopically. It has remained refractory to both internal and local therapy.

The average follow-up of these 34 cases is 19.3 months, ranging from 9 months to 32 months, postoperatively. To date, one patient, in the noncombined-procedure group, died of acute fulminating tuberculous pericarditis (2.9 per cent). The lone case of mortality involves a white man of 40, who had failed to show the anticipated amount of improvement under active medical regime and continued to run a downhill course with repeated bouts of hemoptysis. A Lucite ball plombage was performed and it improved the patient's general condition to a certain extent, but repeated hemoptysis later justified an emergency pneumonectomy. The patient improved and did well for three months, postoperatively, but died in the fifth month postoperatively of acute fulminating tuberculous pericarditis. This patient's sputum was converted directly after resection and remained so until the time of death. There was no death in the combined-procedure group.

As to the present status of the remaining 33 patients who are still alive, Table III will show that 28 patients are well and have been discharged home with negative sputum. Of the 5 patients who are still hospitalized, all are in the combined-procedure group. Three of them are being processed for discharge home as arrested cases of pulmonary tuberculosis, including the one with miliary and meningeal involvement. The remaining 2 still face an uncertain future, one with multiple incisional sinuses as a result of an infected subscapular space, secondary to bronchopleural fistula and empyema, and the other with an infected bronchial stump refractory to local and general treatment. The latter also has a positive sputum. Therefore, out of 17 cases with concomitant procedure, 2 are still hospitalized; one with positive sputum.

TABLE III

| | NUMBER OF PATIENTS | LENGTH OF TIME (POSTOPERATIVELY) | | SPUTUM ANALYSIS (POSTOPERATIVELY) |
|---|---|---|---|---|
| Discharged home | 28 | Within 6 months | 6* | All negative |
| | | Within 9 months | 10 | |
| | | Within 12 months | 12 | |
| | | Within 15 months | 1 | |
| Hospitalized, but in process of being discharged | 3 | (a) 7 months | | (a) Negative 7 months |
| | | (b) 8 months | | (b) Negative 8 months |
| | | (c) 13 months | | (c) Negative last 8 months |
| Hospitalized, but not likely to be discharged soon | 2 | (a) 11 months | | (a) Negative for last 5 months |
| | | (b) 13 months | | (b) Positive to date |

*1 against medical advice; 1 disciplinary discharge.

As to the operative morbidity of this combined procedure, which is reputedly higher than those procedures without concomitant thoracoplasty, we have also failed to find significant difference between the two groups. The following tables (V and VI) are compiled from the anesthesia chart, the progress notes, and the nurses' bedside notes. Table VII will show the computed average of each item from Tables V and VI.

Anesthesia time was used as it is more accurately and constantly recorded than the actual beginning of the operative procedure. It is interesting to observe from the anesthesia chart that the blood pressure usually runs a constant level until actual subperiosteal resection of ribs, second to include the seventh, began. This additional procedure, which usually takes about 15 minutes to 20 minutes, seems to be responsible for a drop on an average of 15 mm. Hg and also a loss of another 200 to 300 c.c. of blood. The pulse rate usually rises on an average of 10 points per minute. These changes in the general condition of the patient, however, do not appear to be of great significance nor serious enough to be called an increase in the operative morbidity. Iverson[19] reported, "Blood is administered during operative procedure in amounts up to 1,500 c.c. and continued after operation up to 2,000 to 2,500 c.c." Our series shows an average of 2,600 c.c. administered in the operating room, and another 700 c.c. after the operation. An average of 1,250 c.c. of blood loss in the drainage bottle was measured in the postoperative period. The drainage usually stops entirely within the first 24 hours. We have not encountered any case of profound shock and in no instance was a drop in blood pressure not immediately responsive to acceleration of the blood transfusion rate in replenishing the blood level. Because of the necessity of the patient to readjust to the loss of the pulmonary vascular bed of one lung, though destroyed in respiratory function, the blood transfusion was given at a slow rate and no incident of pulmonary edema was encountered.

As to the other features in the post pneumonectomy care of the patients, it is gratifying to note that in agreement with Iverson's findings, no bronchoscopic aspiration was found necessary in this series. In fact, this is the most impressive feature of the postoperative period. All patients have an effective cough mechanism and no atelectasis of part of the contralateral lung has occurred. Only a few needed tracheal aspiration, especially those who had had a transient episode of right heart failure. All patients, however, do have some degree of hyperpnea which is not any more pronounced than in those having had a primary pneumonectomy. They usually became comfortable after the first 24 to 48 hours.

Another one of the remarkable features of the postoperative period of such combined procedure is the total "freedom" from concern of a shifted mediastinum and swing. Most of us will agree that in addition to the blood loss and, therefore, shock in pneumonectomized patients, the main complication most dreaded is the mediastinal displacement in the immediate postoperative period. Many a death can be directly traced to this mechanical displacement of the mediastinum with its train of serious, if not fatal, alteration of cardio-respiratory physiology. It is our feeling that in cases with

TABLE IV

| | TOTAL NUMBER | LOW-GRADE FEVER | LOCAL ABSCESS | BRONCHOPLEURAL FISTULA AND EMPYEMA | COR PULMONALE | SPREAD | WOUND INFECTION | DRAINING SINUS | MONALDI WOUND | TUBERCULOUS PERICARDITIS |
|---|---|---|---|---|---|---|---|---|---|---|
| Combined procedure | 3<br>(17.7%) | 0 | 1 | 1* | 2 | 1* | 1* | 1* | 1 | 0 |
| Noncombined procedure | 5 | 2 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 1 |
| Total | 8<br>(23.5%) | | | | | | | | | |

*Same patient.

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| L. R. C.<br>No. 30 | 2 | 36 | Pressure ↓ 20 points<br>Pulse ↑ 10 points<br>From start of operation | 1,615 | 2,500 | 1,500 | 800 | 2 | 7 |
| R. M.<br>No. 18 | 2 | 15 | Pressure ↓ 20 points<br>Pulse ↑ 10 points<br>After 1 hr., 35 min. | 680 | 2,000 | 500 | 1,350 | 2 | 5 |
| A. L. B.<br>No. 16 | 3 | 5 | Pressure ↓ 20 points<br>Pulse ↑ 20 points<br>*After 2 hrs., 30 min.* | 1,810 | 3,000 | 0 | 825 | 1 | 1½ |
| L. R.<br>No. 19 | 2 | 37 | Pressure ↓ 20 points<br>Pulse ↑ 20 points<br>After 1 hr., 50 min. | 1,452 | 2,500 | 0 | 900 | 2 | 2 |
| H. S.<br>No. 20 | 3 | 0 | Pressure ↓ 20 points<br>Pulse ↑ 20 points<br>After 2 hrs., 15 min. | 1,814 | 3,000 | 3,300 | 3,115 | 3 | 2 |
| M. D.<br>No. 28 | 2 | 10 | Pressure ↓ 15 points<br>Pulse ↑ 5 points<br>After 1 hr., 35 min. | 960 | 2,000 | 500 | 750 | 1 | 7 |
| R. L. S.<br>No. 27 | 3 | 0 | Pressure ↓ 20 points<br>Pulse ↑ 20 points<br>After 2 hrs., 20 min. | 3,353 | 5,500 | 2,500 | 935 | 3 | 10 |
| S. L.<br>No. 26 | 2 | 15 | *No change* | 760 | 2,500 | 1,500 | 1,100 | 2 | 3 |
| J. A. J.<br>No. 34 | 2 | 0 | Pressure ↓ 20 points<br>Pulse ↑ 10 points<br>After 1 hr., 30 min. | 593 | 2,000 | 0 | 800 | 1 | 3 |
| Average of 17 patients | 2 | 27 | Pressure ↓ 13 points<br>Pulse ↑ 11 points | 1,310 | 2,588 | 690 | 1,242 | 2 | 3.7 |

TABLE V. CASES WITH CONCOMITANT PROCEDURES

| NAME AND CASE NUMBER | ANESTHESIA TIME | | ALTERATION OF BLOOD PRESSURE AND PULSE DURING OPERATION | BLOOD LOSS DURING OPERATION (C.C.) | BLOOD GIVEN DURING OPERATION (C.C.) | SUBSEQUENT BLOOD GIVEN (C.C.) | BLOOD LOSS IN DRAINAGE BOTTLE (C.C.) | TUBES REMOVED (DAYS) | LENGTH OF DISEASE (YEARS) |
|---|---|---|---|---|---|---|---|---|---|
| | HRS. | MIN. | | | | | | | |
| W. W.<br>No. 11 | 2 | 48 | Pressure ↓ 10 points<br>Pulse ↑ 10 points<br>After 2 hours | 1,320 | 2,500 | 0 | 1,500 | 2 | 2 |
| M. F.<br>No. 21 | 2 | 25 | Pressure ↓ 20 points<br>Pulse ↑ 10 points<br>After 1 hr., 45 min. | 1,148 | 2,000 | 500 | 1,400 | 2 | 4 |
| I. H.<br>No. 3 | 2 | 20 | Pressure ↓ 40 points<br>Pulse stabilized from beginning of procedure | 1,145 | 2,000 | 0 | 0 | 1 | 3 |
| J. M.<br>No. 14 | 2 | 14 | Pressure ↓ 5 points<br>Pulse ↑ 10 points<br>After 1 hr., 45 min. | 1,058 | 2,500 | 0 | 1,950 | 2 | 3 |
| P. W.<br>No. 22 | 1 | 50 | Pressure ↓ 10 points<br>Pulse ↑ 10 points<br>After 1 hr., 10 min. | 760 | 2,000 | 1,000 | 2,000 | 3 | 4 |
| B. B.<br>No. 12 | 2 | 30 | Pressure ↓ 10 points<br>Pulse ↑ 20 points<br>After 1 hr., 45 min. | 1,173 | 2,500 | 0 | 650 | 1 | 1½ |
| L. B.<br>No. 10 | 2 | 10 | Pressure ↓ 15 points<br>Pulse ↑ 10 points<br>After 1 hr., 25 min. | 860 | 2,500 | 0 | 600 | 2 | 1½ |
| L. A.<br>No. 33 | 2 | 25 | Pressure ↓ 15 points<br>Pulse ↑ 10 points<br>After 1 hr., 55 min. | 1,865 | 3,000 | 500 | ? | 1 | 3 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| B. B.<br>No. 6 | 3 | 10 | Pressure ↓ 30 points<br>Pulse stable<br>After 1 hr., 30 min. | 3,635 | 6,000 | 500 | 900 | 1 | 8 |
| E. H.<br>No. 32 | 1 | 50 | No significant continuous drop | 1,908 | 3,500 | 1,000 | 1,500 | 2 | 6 |
| E. M. C.<br>No. 1 | 2 | 0 | No change | 858 | 1,500 | Not recorded | Not recorded | 2 | 1 |
| H. B.<br>No. 15 | 3 | 0 | No change | 1,650 | 3,000 | 500 | 850 | 1 | 2 |
| C. C.<br>No. 2 | 2 | 30 | Pressure ↓ 30 points<br>Pulse ↑ 10 points<br>After 1 hr., 45 min. | 1,351 | 2.500 | 500 | 800 | 1 | 2 |
| B. J.<br>No. 25 | 2 | 15 | Pressure ↓ 10 points<br>Pulse ↑ 5 points<br>After 1 hr., 45 min. | 1,551 | 3,000 | 0 | 975 | 2 | 3 |
| R. D.<br>No. 23 | 2 | 30 | No change | 1,185 | 2,500 | 0 | 625 | 1 | 6½ |
| S. C.<br>No. 24 | 2 | 0 | Pressure ↓ No change<br>Pulse ↑ 30 points<br>After 1 hr. | 990 | 2,500 | 0 | 500 | 1 | 7 |
| Average<br>13 patients<br>4 patients | 1<br>2 | 8 ><br>24 > | Pressure ↓ 14 points<br>Pulse ↑ 8 points | 1,498 | 2,790 | 409 | 1,039 | 2 | 4.4 |

TABLE VI. CASES WITHOUT CONCOMITANT THORACOPLASTY

| NAME AND CASE NUMBER | ANESTHESIA TIME | | ALTERATION OF BLOOD PRESSURE AND PULSE DURING OPERATION | BLOOD LOSS DURING OPERATION (C.C.) | BLOOD GIVEN DURING OPERATION (C.C.) | SUBSEQUENT BLOOD GIVEN (C.C.) | BLOOD LOSS IN DRAINAGE BOTTLE (C.C.) | TUBES REMOVED (DAYS) | LENGTH DISEASE (YEARS) |
|---|---|---|---|---|---|---|---|---|---|
| | HRS. | MIN. | | | | | | | |
| E. B.<br>No. 9 | 2 | 10 | No change | 1,366 | 2,500 | 500 | 1,450 | 2 | 9 |
| A. T.<br>No. 5 | 2 | 30 | Pressure ↓ 20 points<br>Pulse ↑ 5 points<br>After 2 hrs. | 1,271 | 2,000 | 500 | 900 | 1 | 3½ |
| M. H. T.<br>No. 4 | 2 | 25 | Pressure ↓ 25 points<br>Pulse ↑ 10 points<br>After 2 hrs. | 1,603 | 2,500 | 0 | 500 | 2 | 7 |
| H. B.<br>No. 13 | 2 | 10 | Pressure ↓ 40 points<br>Pulse ↑ 20 points<br>From beginning of operation | 1,715 | 3,000 | 1,000 | 1,475 | 2 | 2½ |
| M. Y.<br>No. 8 | 2 | 10 | Pressure ↓ 30 points<br>Pulse ↑ 20 points<br>From beginning of operation | 729 | 2,000 | 0 | 600 | 2 | 1 |
| T. W.<br>No. 29 | 2 | 6 | Pressure ↓ 20 points<br>Pulse ↑ 20 points<br>After 1 hr., 25 min. | 812 | 2,000 | ? | ? | ? | 5 |
| K. S.<br>No. 17 | 1 | 40 | Pressure ↓ 10 points<br>Pulse stable<br>Throughout procedure | 606 | 2,000 | 1,000 | None recorded | 2 | 4 |
| B. D.<br>No. 31 | 3 | 0 | Pressure ↓ 20 points<br>Pulse ↑ 20 points<br>After 2 hrs., 30 min. | 2,450 | 3,500 | 500 | 2,700 | 3 | 4 |
| J. C.<br>No. 7 | 2 | 30 | Pressure ↓ 20 points<br>Pulse ↑ 20 points<br>After 1 hr., 45 min. | 1,785 | 3,500 | 0 | 775 | 3 | 2 |

the decostalized chest wall, allowing the shoulder blade structures to fall in, affords a much more solid support to the mediastinum than would a compressible cushion of air in an empty pleural space, as in the case of primary pneumonectomy. This support is reinforced further by a tight pressure dressing to eliminate any paradoxical motion in that region. Allowing for the fact that there is no total obliteration of pleural space by the described thoracoplasty, it is believed that the thin layer of coagulum in the residual space still provides a less compressible background. In all the 12 to 24 hour postoperative chest films in this series, the pneumonectomized "lung field" shows total opacity with no air pocketing in spite of the drainage tube. We believe that this firm support to the mediastinal structures prevents any dangerous degree of swinging and displacement. We also have never found it necessary to readjust the intrapleural pressure for correction. In fact, we feel that it is this more stabilized mediastinum that improves the efficacy of the cough mechanism, as less of its expulsive force is dissipated through a swing of the mediastinal structures. This more stabilized mediastinum and a pleural space that is greatly reduced by the concomitant thoracoplasty are believed to allow adequate underwater drainage without the danger of overdecompression and shifting of the mediastinum to a dangerous degree, as in the case of primary pneumonectomy.

It would seem, therefore, in the patients with concomitant pneumonectomy and thoracoplasty that there is no specific problem in the immediate postoperative period except for the blood replacement. We are impressed by the relatively benign postoperative course of these patients in the critical postoperative period. It is, therefore, our feeling that this concomitant thoracoplasty improves or reduces the postoperative morbidity rather than otherwise.

## DISCUSSION

It is not the intention of this paper to recommend pneumonectomy with concomitant thoracoplasty as a procedure to be applied to all routine cases of tuberculosis which require total lung resection. However, it is the purpose of this article, on a comparative study of 17 cases of combined procedure and 17 comparable cases without combined procedure, to re-evaluate the degree of selectivity for application of this concomitant procedure. Our experience seems to corroborate the statement made by Iverson that the procedure does not materially increase the operative time, blood loss, or over-all morbidity. The efficacy of cough in the postoperative period is impressively enhanced over that following primary pneumonectomy. The "freedom" of concern about mediastinal swing and displacement is also a striking feature in the immediate postoperative period. As to the effect of thoracoplasty on the breathing capacity of the remaining lung, we have not come across any patient who suffers from increasing impairment. In patients who have a longer period of follow-up, there was no evidence of scoliosis (Fig. 5) and, thus, no decrease of ventilatory effort with the passage of time is anticipated. This is primarily attributable to the preservation of relatively long anterior stumps of ribs and

TABLE VII

| | ANESTHESIA TIME | | ALTERATION OF BLOOD PRESSURE AND PULSE DURING OPERATION | BLOOD LOSS DURING OPERATION (C.C.) | BLOOD GIVEN DURING OPERATION (C.C.) | SUBSEQUENT BLOOD GIVEN (C.C.) | BLOOD LOSS IN DRAINAGE BOTTLE (C.C.) | TUBES REMOVED (DAYS) |
|---|---|---|---|---|---|---|---|---|
| | HRS. | MIN. | | | | | | |
| Combined procedure | 27 | 2 | Pressure ↓ 15 points<br>Pulse ↑ 10 points | 1,310 | 2,588 | 690 | 1,240 | 1.8 |
| Separate procedure | 24 | 2 | Pressure ↓ 14 points<br>Pulse ↑ 8 points | 1,500 | 2,790 | 500 | 1,110 | 2.0 |

due to the ineffectual coughing mechanism, in the presence of a flail chest wall, to evacuate secretions in the ipsilateral lung tissue. In combined lobectomy and thoracoplasty, when usually no more than a five-rib thoracoplasty is performed, the subscapular space is not obliterated by the falling in of the shoulder blade structure and thus the paradoxical motion of the decostalized chest wall persists under the scapula. Stead[28] has demonstrated the undesirable features of such combined procedure very convincingly in his physiologic studies. In the case of pneumonectomy, where there is no lung tissue left in the pleural space with a flail and decostalized chest wall, the danger of such complication, therefore, does not exist.

In view of our experience with this combined procedure of pneumonectomy and thoracoplasty, we would like not only to corroborate the feelings of Iverson and Samson, but also to hope for wider application of this procedure. It is felt that, except in cases with serious contraindication, such as in the extreme of life, severe degenerative diseases, or blood transfusion reaction, etc., consideration of this procedure in appropriate cases should be seriously entertained. As to its usefulness in pneumonectomy for diseases other than tuberculosis, reevaluation should also be made with renewed interest. Since Evarts Graham did the first successful pneumonectomy and concomitant thoracoplasty for bronchogenic carcinoma of the lung, this procedure has not been given the proper amount of evaluation and consideration to have it stigmatized as too radical an operation. It is felt that this procedure should be of help in the problem of the remaining space until such time, however remote, when the present difficulty in prosthesis will be resolved. The fact that tuberculous patients are conceded to be more likely to have a contracted blood volume, subnormal liver function, and be generally weaker than a person who has only short-term disease, such as carcinoma or lung abscess, one cannot help but wonder whether the satisfactory results in this series, however small it is, could not be duplicated, if not bettered, in nontuberculous patients. At least this procedure deserves the consideration of a surgeon who might find occasion to employ it.

## CONCLUSION

A comparative study has been made of 17 cases of combined pneumonectomy and thoracoplasty and 17 cases of separate procedure, done consecutively over a period of 21 months. The two groups were compared first and found to have essentially similar composition as regards to race, sex, age groups, extent of disease, activity of lesions ascertained from resected specimen, length of disease, and length of preoperative antituberculosis drug therapy. It was found that from these two comparable groups, the final results as to sputum conversion, arrest of disease, discharge status, and mortality rate, are closely akin and also compare favorably with other series of primary pneumonectomy. It was also found that the operative and immediate postoperative morbidity rate were not as high as reputed to be, nor out of order when compared with that of other series. In fact, the increase in efficacy of cough mechanism and general lack of complications secondary to mediastinal displacement in the immediate postoperative period are especially impressive. It is, therefore, [illegible] un [illegible] nted th the transverse processes. In cases of a potentially infected pleural space, as in the patient who had a Monaldi tract, we were particularly gratified by the lack of empyema formation, a result, made possible, at least partially, by the safe and adequate drainage of the residual pleural space with the concomitant thoracoplasty. As stated, such space could be drained until all oozing and weeping had ceased, carrying with it most of the infected organisms, without the danger of overdecompression with shifting of mediastinum, as in case of primary pneumonectomy. In addition, the findings from the analysis of the group with combined procedure indicated that the operative and postoperative morbidity and mortality compares favorably, not only with the patients who had the separate procedure, but with other series of primary pneumonectomy as well. Samson,[27] in 1951, mentioned 15 cases of pneumonectomy and concomitant thoracoplasty "with no immediate postoperative complication of major concern." It is, therefore, felt that possibly the selectivity of this combined pneumonectomy and thoracoplasty should be less than formerly believed to be necessary. It is also felt that the notoriety given the combined procedure in the literature, as being a very risky operation, accompanied with high morbidity if not mortality, is not fully justified. It is unfortunate that a distinction has not always been made between operative morbidity and mortality of the combined procedure of pneumonectomy and thoracoplasty and that of lobectomy and concomitant thoracoplasty. The lung tissue in a freshly decostalized pleural cavity is more likely to be involved in complications such as atelectasis, poor re-expansion of lung, hemothorax, infection of pleural space, etc. This is

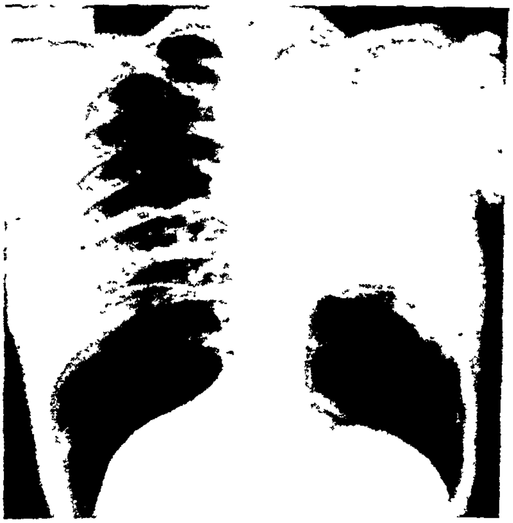


Fig. 4.

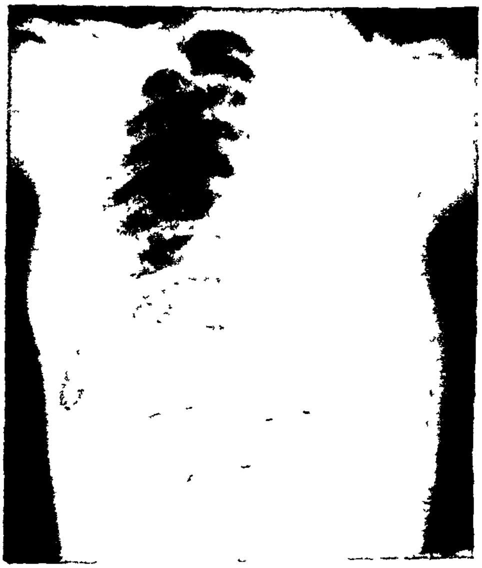


Fig. 5.

Fig. 4.—Case No. 33. L. A., a 23-year-old white woman, was admitted March 2, 1950. ar advanced, C. She received various combinations of streptomycin, INAH, and PAS, and neumoperitoneum for three years. A combined procedure was performed on July 29, 1953, rith uneventful recovery. Sputum was positive for the last time on Nov. 3, 1952, and has remained egative since. Specimen shows destroyed lung with bronchiectasis, endobronchitis, and active uberculosis granulation tissue. She was discharged home June 24, 1954.

Fig. 5.—Case No. 33. L. A., same patient. Film taken prior to discharge June 24, 1954, 11 months postoperatively. It shows the lack of gross scoliosis.

# EXPERIMENTAL INTERATRIAL SEPTAL DEFECT HEMODYNAMIC AND ELECTROCARDIOGRAPHIC STUDIES AT NORMAL TEMPERATURE AND DURING ARTIFICIAL HIBERNATION

VIRGILIO A DE CARVALHO PINTO M D *
SÃO PAULO, BRAZIL

THE surgical repair of the interatrial septal defect (IASD), a fairly common malformation, has been carried out successfully in recent years Such successful clinical results have been largely due to experimental studies Lack of data on several features related to IASD, such as the hemodynamic and electrocardiographic changes derived from the creation and closure of the defect, has led us to the present investigation This paper is a report of our experience with the physiologic and surgical aspects of the creation and closure of the IASD in the dog, under different conditions

Creation and closure of a IASD in the same operative sitting, under normal temperature conditions and during artificial hibernation, were carried out in 50 animals The results obtained under strictly homogeneous conditions were submitted to statistical analysis

## MATERIAL AND METHODS

Healthy, adult, male mongrel dogs, weighing 15 to 18 Kg, were divided into two groups (1) Twenty animals were operated on under normal temperature conditions, (2) thirty animals were operated on during artificial hibernation (hypothermia obtained by pharmacologic means plus refrigeration—Laborit) In both groups, the blood flow was temporarily occluded (40 to 50 seconds) and the IASD created under direct vision Closure of the defect in Group I was carried out according to the technique used by Gross (1952) and, in Group II, under direct vision and interruption of blood flow (6 minutes)

Intracardiac pressure curves in the left and right atria and right ventricle as well as electrocardiographic tracings in the classic, unipolar limb and direct epicardial leads were recorded in all animals, before creation of the defect, while it was open, and after its closure In Group II, electrocardiograms were also taken before opening the chest and after surgical repair, with closed chest and return to initial temperature after rewarming

*Anesthesia in Group I*—Operations were performed under intravenously administered Nembutal anesthesia (0 33 gram per Kg of body weight)

---

Abridgment of thesis submitted to the Medical School of the University of São Paulo (Brazil) for the degree of Privat Dozent

Received for publication April 9 1956

* Assistant Professor at the Department of Surgery and Experimental Surgery Medical School of the University of Sao Paulo (Director Prof Silv

the highly selective use of this concomitant pneumonectomy and thoracoplasty should be re-evaluated as to its range of applicability, not only in tuberculous patients, but in nontuberculous patients as well.

## REFERENCES

1. Freedlander, S. C.: *Lobectomy in Pulmonary Tuberculosis,* J. THORACIC SURG. 5: 132, 1935.
2. Jones, J. C., and Dolley, F. S.: Lobectomy and Pneumonectomy in Pulmonary Tuberculosis, J. THORACIC SURG. 8: 315, 1939.
3. Janes, R. M.: Total and Partial Pneumonectomy in the Treatment of Pulmonary Tuberculosis, J. THORACIC SURG. 14: 3, 1945.
4. Sweet, R. H.: *Lobectomy and Pneumonectomy in the Treatment of Pulmonary Tuberculosis,* J. THORACIC SURG. 15: 373, 1946.
5. Overholt, R. D., Woods, F. M., and Wilson, N. G.: Segmental Resection in Tuberculosis, *Dis. Chest 23: 255, 1953.*
6. Chamberlain, J. M., and Klopstock, R.: Further Experiences With Segmental Resection in Tuberculosis, J. THORACIC SURG. 20: 843, 1950.
7. Churchill, E. D., and Belsey, R.: *Segmental Pneumonectomy in Bronchiectasis,* Ann. Surg. 109: 481, 1939.
8. Blades, B.: Conservation of Lung Tissue by Partial Lobectomy, Ann. Surg. 118: 353, 1934.
9. Rasmussen, R. A., Adams, W. E., and Hrdina, L. S.: *Should the Pleural Space Be Reduced* in Size in the Resection of Lung Tissue? Surgery 10: 85, 1941.
10. Grindlay, J. H., and Claggett, O. T.: A Plastic Sponge Prosthesis for the Use After Pneumonectomy. Preliminary Report of an Experimental Study, Proc. Staff. Meet., Mayo Clin. 124: 538, 1949.
11. Charbon, B. C., and Adams, W. E.: A Study to Determine Effect of Prevention of Overdistention of Lung Tissue Remaining on the Elevated Right Ventricular Pressures, Following Resection of Lung Tissue in Dogs, J THORACIC SURG. 23: 341, 1952.
12. Levine, S., Grow, J. B., Hurst A. and Perlmutter A.: Experimental Study of Plastic Synthetics in Thoracic Surgery, Dis. Chest 20: 126, 1951.
13. Grindley, J. H., and Waugh, J. M.: Plastic Sponge Which Acts as a Framework for Living Tissue, Arch. Surg. 63: 288, 1951.
14. Brantigan, O. C., and Rigdon, H. L.: Pulmonary Prosthesis After Pneumonectomy, J. THORACIC SURG. *20: 109, 1950.*
15. Gale, J. W., Currerri, A. R., Young, W. P., and Dickie, H. A.: Plastic Sponge Prosthesis After Resection, J. THORACIC SURG. 24: 587, 1952.
16. Hertzog, M.: Une technique d'effacement de la cavite restante après pneumonectomie, Pouman 8: 319, 1952.
17. Metras and Gregoire: Traitment de la cavite libre apres pneumonectomie, Pouman 8: 313, 1952.
18. Iverson, R. K., and Skinner, H. L.: *Pneumonectomy Followed by Immediate Thoracoplasty.* Preliminary Report of Three Cases, Pub. Health Rep. 64: 140, 1949.
19. Iverson, R. K., and Skinner, H. L.: Pneumonectomy Followed by Immediate Thoracoplasty, J. THORACIC SURG. 19: 491, 1950.
20. Samson, P. C., Dugan, D. J., and Harper, H. P.: Upper Lobectomy and Concomitant Thoracoplasty in Pulmonary Tuberculosis, California Med. 73: 547, 1950.
21. Conklin, W. S., Tuhy, J. E., and Grismer, J. T.: Thoracoplasty Combined With Resection for Pulmonary Tuberculosis, J. THORACIC SURG. 22: 271, 1951.
22. Cruickshank, G., and Papamichael, E.: Pneumonectomy With Immediate Thoracoplasty in the Treatment of Pulmonary Tuberculosis, Thorax 6: 369, 1951.
23. Shek, J. L., and Corpe, R. F.: Comparative Study of Postresection Morbidity in Cases With and Without Preresection Thoracoplasty, J. THORACIC SURG. 32: 92, 1956.
24. O'Brien, W. B., Wilson, N. J., Armada, O., and Vindzberg, A.: Total Surgical Statistics in the Treatment of Pulmonary Tuberculosis, Am. Rev. Tuberc. 68: 874, 1953.
25. McBurney, R. P., Hedberg, G. A., Jampolis, R. W., Clark, P. LeM., III, and Krueger, V. R.: Complication Associated With Resective Operations for Pulmonary Tuberculosis, J. THORACIC SURG. 27: 330, 1954.
26. Baffes, T. G., Schlotterbeck, P., and Decamp, P. T.: A Comparison of Thoracoplasty and Resection in Pulmonary Tuberculosis, J. THORACIC SURG. 27: 349, 1954.
27. Samson, P. C.: Discussion of paper by Conklin, Tuhy, and Grismer.[21]
28. Stead, W. W.: Physiologic Studies Following Thoracic Surgery. II. Immediate Effects of Upper Lobectomy Combined With a Five-Rib Thoracoplasty, J. THORACIC SURG. 25: 194, 1953.

inferior venae cavae were dissected and mobilized. Cotton thread sutures (No. 3-0) were placed around them. Two retraction silk sutures were placed on the atrial wall and a Satinsky clamp was placed on the same. The mean temperature of dogs submitted to hibernation was 27.5° C.

*Creation of IASD Under Direct Vision.*—Interruption of blood flow in the superior and inferior venae cavae (Fig. 1). Section and removal of the greater part of the septum, creating a large defect through which the index finger could be introduced with ease, 1.5 cm. in diameter (Fig. 2). Circulation was restored after 40 to 50 seconds of interruption. The atrial wall was closed with a running suture of No. 3-0 silk. The mean temperature of hibernated dogs was 26.3° C.

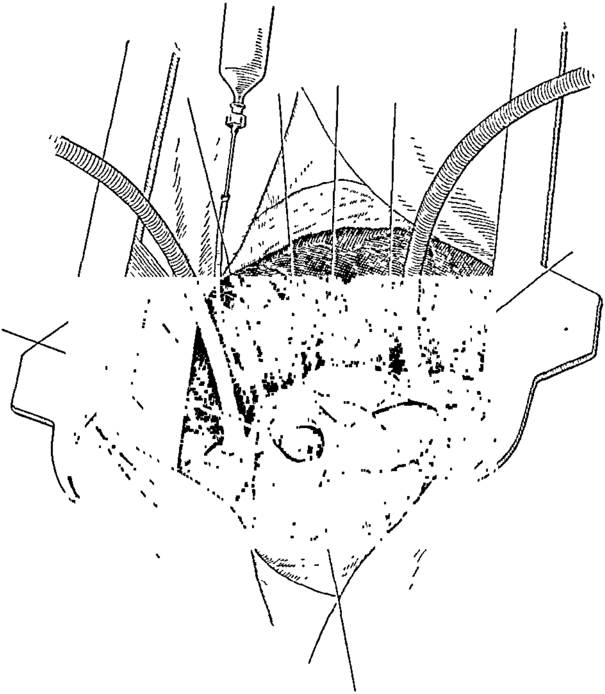


Fig. 2.—Defect created in the septum

*Closure of IASD.*—Group I. The septal defect was closed according to Gross (Fig. 3) in the animals operated on at normal temperature. The atrium remained open from 6 to 8 minutes.

Group II. The septal defect was closed under direct vision in the hi [illegible] nated dogs. After reopening the original atrial [illegible] of [illegible]

*Anesthesia and Hibernation in Group II.*—Intramuscular Amplictil and Phenergan (Rhodia), 0.8 mg. per Kg. of body weight was used, followed by slow intravenous administration of Amplictil and Phenergan forty-five minutes later (0.8 mg. per Kg. of body weight), dissolved in 20 c.c. of saline solution. Slow, intravenous administration of Pentothal sodium (2.5 per cent), 0.003 gram per Kg. of body weight. Endotracheal intubation. The animal was immersed in water containing ice cubes, until rectal temperature dropped to 29° C., which occurred after 20 to 30 minutes. Artificial respiration was maintained with Takaoka's apparatus connected to an oxygen pump. There was intravenous administration of procaine 4 per thousand, plus Amplictil and Phenergan throughout the procedure.

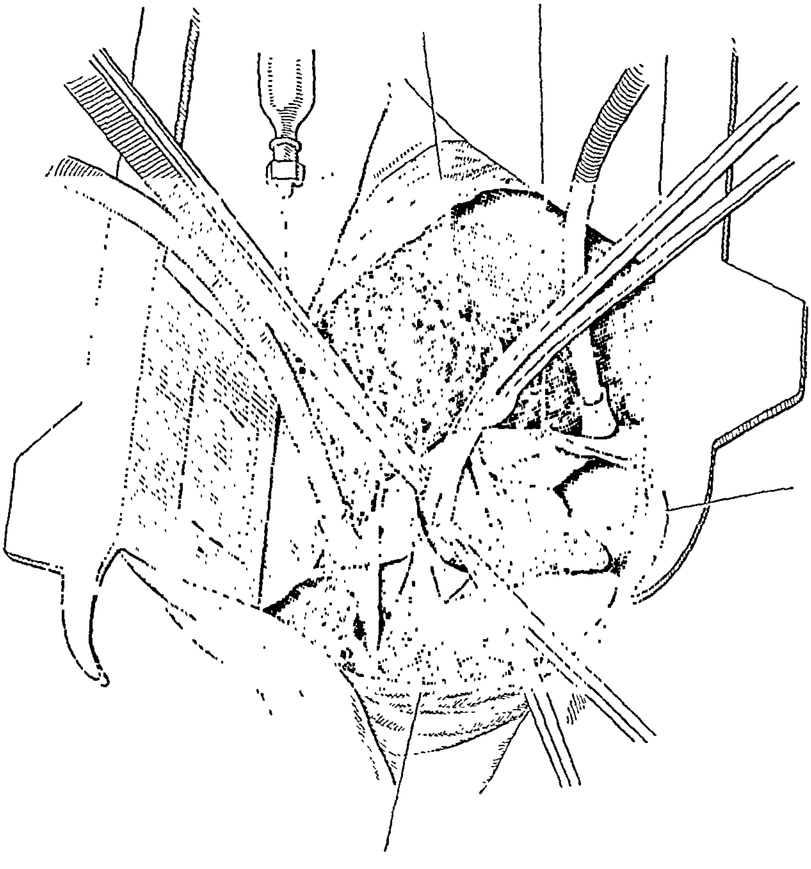


Fig. 1.—Interruption of the venae cavae. Satinsky's clamp withdrawn. Removal of interatrial septum.

After surgery, rewarming was accomplished by placing the animal in a bath at 40° C., until temperature returned to normal level (approximately three hours).

SURGICAL PROCEDURES

Incision was through the fourth right interspace. Five cubic centimeters of 1 per cent procaine was injected into the pericardium. The pericardium was widely opened ventrally to the phrenic nerve and retracted by three sutures. Dissection and ligature of the azygos vein were carried out. The superior and venae cavae, pulmonary artery and aorta up to the origin of the coronary arteries (Figs. 4 and 5). The septal defect was closed under direct vision, with Atraumatic needle, No. 3-0 silk, by three stitches. Cardiac and thoracic cavities were flooded with saline solution. Suture of atrial wall with separated stitches, after restoration of circulation. There was interruption of circulation in the venae cavae for 6 minutes (1.5 for suture of defect). The mean temperature of the hibernated dogs was 25.7° C.

Standard closure of the chest. The animals were sacrificed the day following the operation.

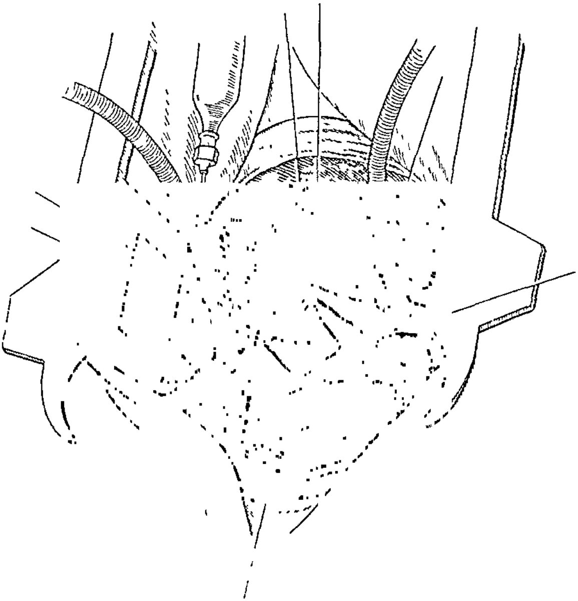


Fig. 5—Complete closure of interatrial septal defect.

## RECORDS OF INTRACARDIAC PRESSURES

Pressures in right and left atria and right ventricle were recorded by means of the Sanborn electromanometer, model 121-A, during the three surgical stages; prior to creation of IASD, when it was open, and after its closure.

For the atrial pressure measurement, the average and instantaneous pressures were considered. The former was obtained by planimetric integration and the latter at the extreme values of the following waves (Fig. 6):

Wave A—positive wave, corresponding to the elevation of the intra-atrial pressure, determined by the contraction of atrial walls.

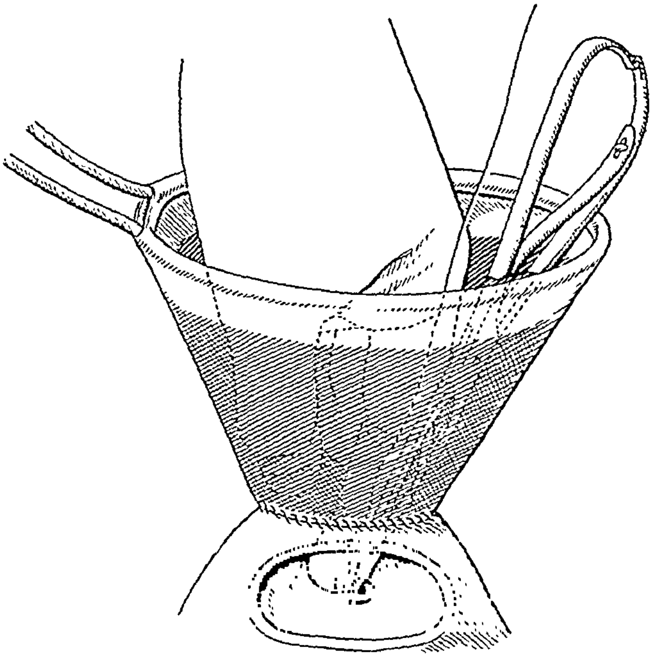


Fig. 3.—Closure of interatrial septal defect, according to Gross; first stitch.

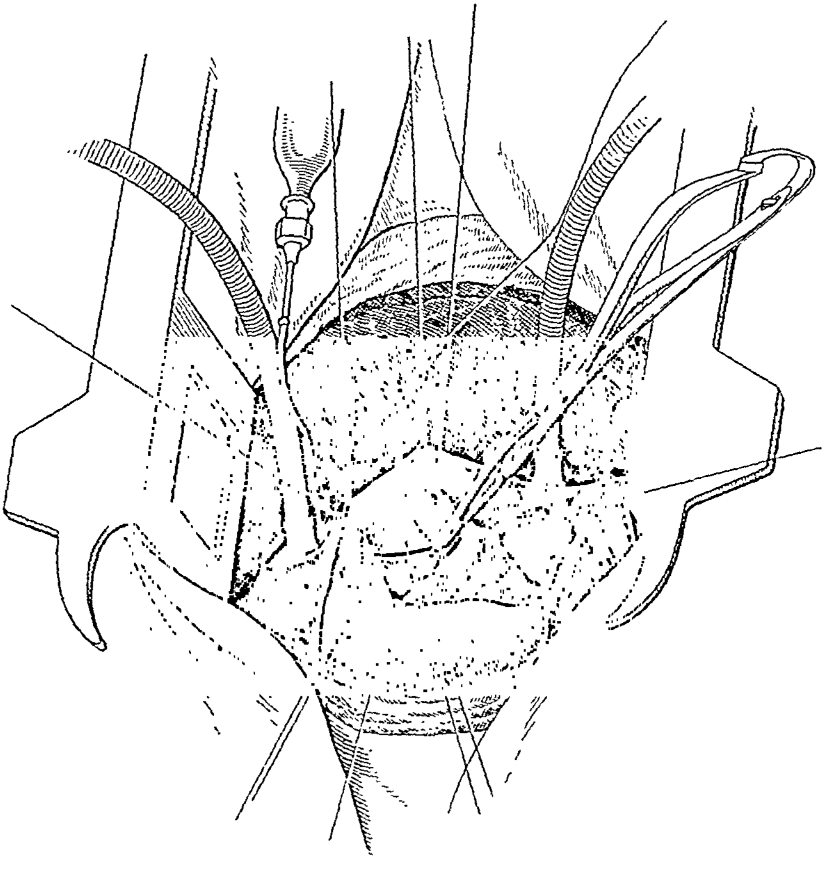


Fig. 4.—Closure of interatrial septal defect under direct vision.

Wave B—positive wave, determined by the increase of intra atrial pressure at the moment in which the atrioventricular valves are closed

Wave D—positive wave which expresses atrial filling, during the period at which the atrioventricular valves are closed

Wave A′—negative wave corresponding to the end of the atrial systole

Wave Y—negative wave which follows the D wave and precedes the A wave, expresses the degrees of atrial emptying, after opening the atrioventricular valves

For the ventricular pressure measurement, the average and instantaneous pressures were taken into consideration The first was obtained by planimetric integration and the latter at the extreme values of the following waves

$Ps_1$—positive wave of the ventricular curve, corresponding approximately to the end of the isometric contraction

$Ps_2$—positive wave following a negative deflexion after $Ps_1$

$Pd_1$—negative wave after the isometric relaxation

$Pd_2$—negative wave at the end of the ventricular systole

The nomenclature used was suggested by Borges (1953, 1954, 1955), based on the studies of Wenckebach and Winterberg (1927) for the atrial pressures and on the nomenclature adopted by the Laboratory of Hemodynamics of the National Institute of Cardiology of Mexico for the ventricular pressures

ELECTROCARDIOGRAPHIC STUDY

Electrocardiographic tracings were recorded by means of the Viso Cardiette Sanborn apparatus model 52, under the usual conditions of film speed and amplitude padronization Classic leads ($D_1$, $D_2$, $D_3$), unipolar limb leads of Goldberger (aVR, aVL, aVF), and direct epicardial leads were taken with the animal in the same position (left lateral decubitus)

Sequence of tracings

a *Animals operated on at normal temperature* Classic and unipolar limb leads of the anesthetized animal, after opening of the chest, epicardial leads before creation of IASD, epicardial leads after creation of the IASD, classic and unipolar limb leads after creation of IASD, epicardial leads after closure of IASD, classic and unipolar limb leads after closure of IASD, with open chest

b *Animals operated on under artificial hibernation* Classic and unipolar limb leads of the animal at normal temperature, classic and unipolar limb leads of the hypothermic animal, after opening the chest epicardial leads before creation of IASD, epicardial leads after creation of IASD, classic and unipolar limb leads after creation of IASD, epicardial leads after closure of IASD, classic and unipolar limb leads after closure of IASD, classic and unipolar limb leads after rewarming to normal temperature

The direct epicardial leads were taken according to Barbato (1952) Tracings were studied according to classic rules of electrocardiography and, of

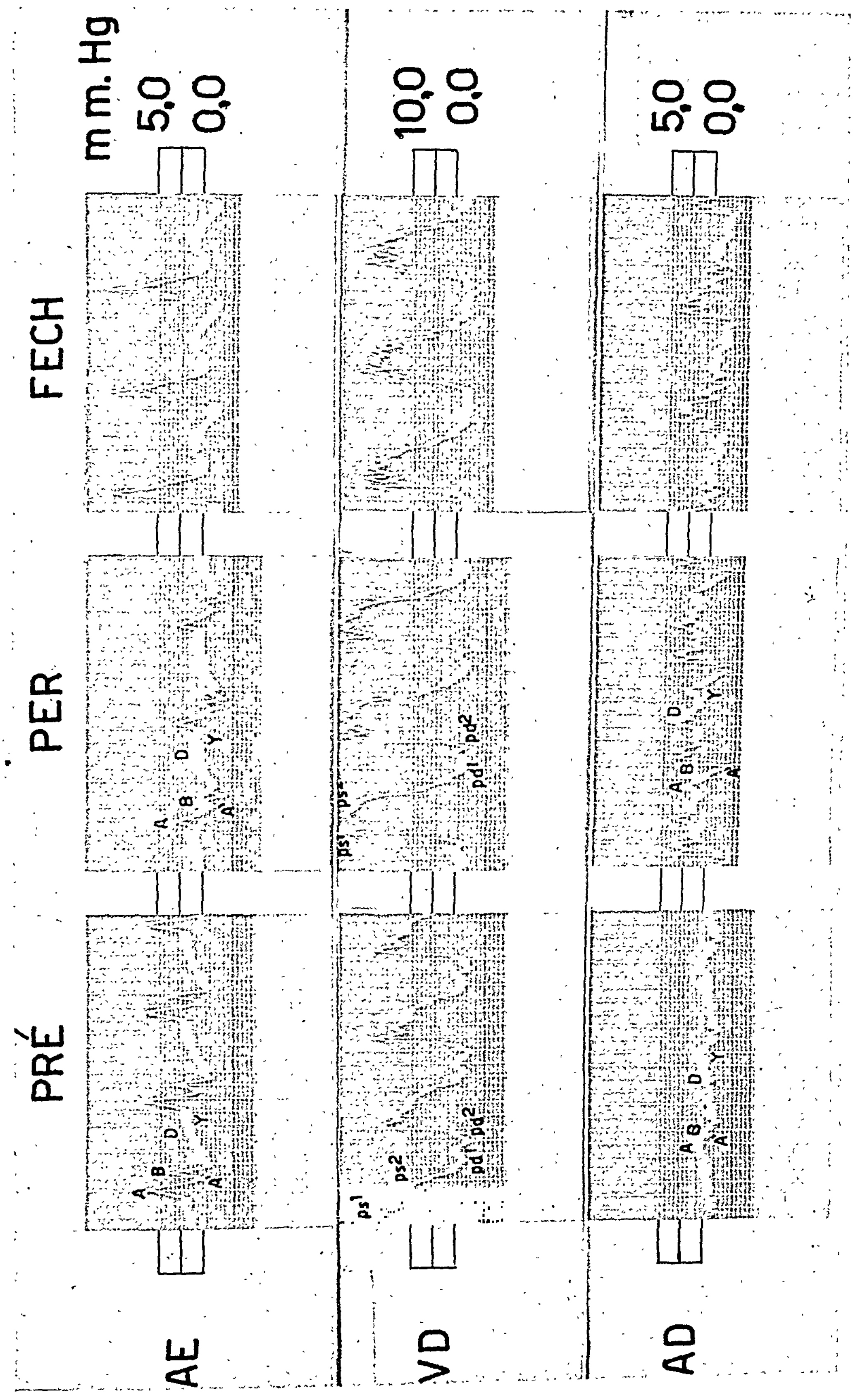


Fig. 6.— Records of intracardiac pressures of Observation No. 30.

FECH-PER—changes caused by the closure of the IASD to the values observed while it was open.

FECH-PRÉ—relations between values observed after closure and those prior to creation of IASD.

AQ-FECH—modifications produced by rewarming to the values observed after closure of IASD.

AQ-NM—relations between values observed after rewarming and those of normal temperature.

The percentage variations were obtained by the following relations: PRÉ-NM/|NM|; PER-PRÉ/|PRÉ|; FECH-PER/|PER|; FECH-PRÉ/|PRÉ|; AQ-NM/|NM|. The denominator represents the absolute value.

The several items studied, concerning hemodynamic and electrocardiographic findings, are presented in tables and analyzed according to their maximal and minimal values and arithmetic mean, standard deviation, test of mean difference and distribution of cases, according to one of the three following conditions: positive difference (increased); negative difference (decreased); null difference (unaltered).

Percentage variations were used and, the increase or decrease of the values above 10 per cent or below—10 per cent, respectively, was considered.

## RESULTS

The results refer to data obtained only under sinusal rhythm:

1. Intracardiac pressures of dogs operated on under normal temperature conditions (Table I, Fig. 7).
2. Intracardiac pressures of dogs operated on during artificial hibernation (Table II, Fig. 8).
3. Electrocardiographic tracings of dogs operated on under normal temperature conditions (Table III, Fig. 9).
4. Electrocardiographic tracings of dogs operated on during artificial hibernation (Table IV, Fig. 10).

## DISCUSSION

Several investigators have reported different techniques for the production of the interatrial septal defect, some by blind techniques, others under direct vision, or with the aid of special instruments. The production of IASD brought us the first technical problems. As the defect should be relatively large, the greater part of the septum had to be removed. It appeared desirable, therefore, to create the lesion under direct vision, according to Swan and co-workers (1950). Standardization of the operative procedure enabled us to create the defect in a relatively short time (40 to 50 seconds), with good results.

After recording the intracardiac pressures and the electrocardiographic tracings during the presence of the IASD, closure was performed.

The experimental approach to direct vision surgery of the heart is being studied at the present time by several authors, with the use of extracorporeal circulation, hypothermia, and extracorporeal circulation [illegible]ted [illegible] hypothermia.

each observation, the following items were analyzed: rhythm, frequency, P wave (duration, amplitude, P angle, slurring and notch), P-R interval, QRS complex (duration, amplitude, QRS angle, slurring and notch), electrocardiographic position of the heart, Q-T interval and R/S relation of direct ventricular epicardial tracings.

To remove the influence of frequency on the P-wave duration values, P-R interval, duration of the QRS complex and on the Q-T interval, $P_c$, P-$R_c$, $QRS_c$ and Q-$T_c$ were studied, respectively, according to the following formulas:

$P_c$, that is, the duration of the "corrected" P wave, according to the formula P-$P_c$ (R-R),$^{0.12196}$ modified from Fujiwara (1939). Before using it, a statistical test was made.[*] This demonstrated its adaptability to our cases.

P-$R_c$, that is, the "corrected" P-R interval, obtained by the same formula used for P, also adaptable to our data.

$QRS_c$, that is, the duration of the "corrected" QRS complex, calculated according to the formula QRS = $QRS_c$ R-R suggested by the formula used for man (Albers and Bedbuhr, 1941), and which was adaptable to our material.

Q-$T_c$, that is, the "corrected" Q-T interval, calculated by the formula of Bazett, Q-T = Q-$T_c$ R-R, and which was adaptable to our cases.

The QRS amplitude was studied, taking into consideration the electrocardiographic position of the heart, and determined according to Sodi-Pallares (1951).

The changes of the RS-T segment are presented only in the hibernated group, as the changes found in the series of animals operated upon at normal temperature do not differ from those commonly found with the simple exposure of the heart to air and room temperature.

The data obtained in the hemodynamic and electrocardiographic records are presented with the following abbreviations which designate the time at which they were performed:

PRÉ—before creation of IASD
PER—after creation of IASD
FECH—after closure of IASD

In the hibernation group:

NM—prior to artificial hibernation.
AQ—after rewarming.

The changes observed in the several experimental phases are expressed by the differences between the corresponding values and percentage variations, in the following way:

PRÉ-NM—changes produced by hibernation to the values of normal temperature.
PER-PRÊ—changes caused by the production of IASD to the values observed before the creation of the defect.

---

[*] From the equation $y = k\,x^c$, where $y = P$ and $x =$ R-R, the hypothesis of $c = 0.122$ was tested, calculating

$$F = \frac{(N-2)\,\{N\Sigma(\log x)\ (\log y) - (\Sigma \log x)\ (\Sigma \log y) - c[N\Sigma(\log x)^2 - (\Sigma \log x)^2]\ \}^2}{[N\Sigma(\log y)^2 - (\Sigma \log y)^2]\ [N\Sigma(\log x)^2 - (\Sigma \log x)^2] - [N\Sigma \log x \log y - (\Sigma \log x)\ (\Sigma \log y)]^2}$$

in which F has the distribution F of Snedecor for 1 and N-2 degrees of liberty.

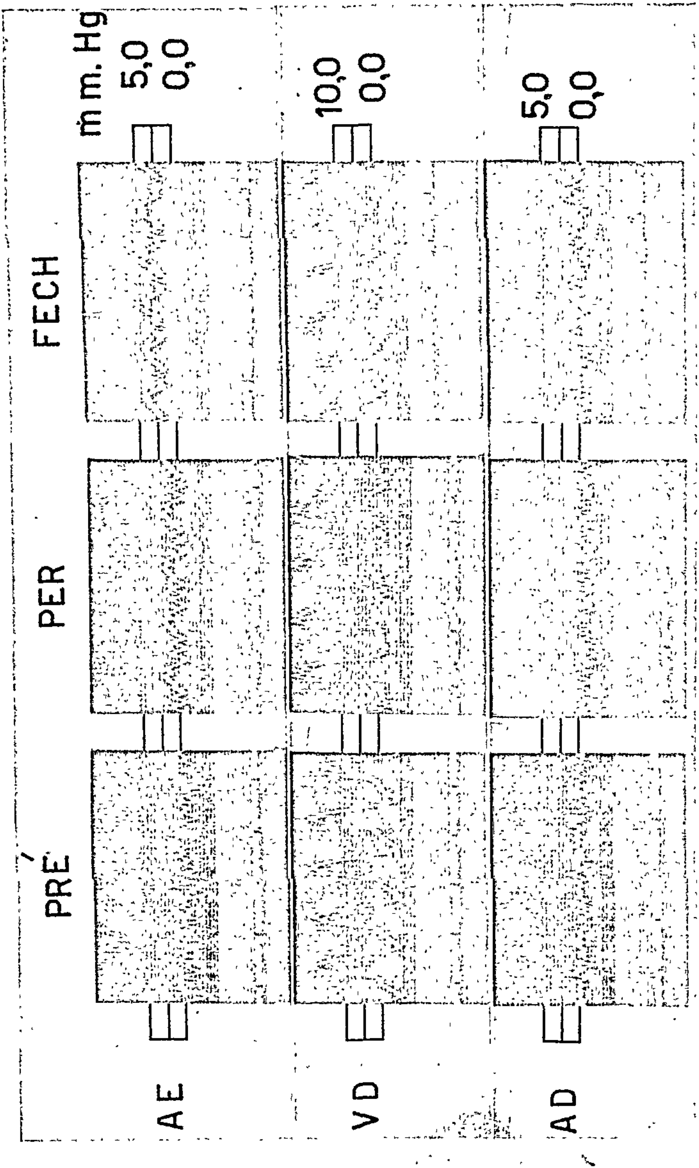


Fig. 7.—Records of intracardiac pressures of Observation No. 7.

TABLE I. SYNTHESIS OF THE RESULTS OF INTRACARDIAC PRESSURES OF THE CASES UNDER SINUSAL RHYTHM

(Animals operated on at normal temperature, mm. Hg)

| | | AE | | | | | | AD | | | | | | VD | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | MEAN PRESSURE | A | B | D | A' | Y | MEAN PRESSURE | A | B | D | A' | Y | MEAN PRESSURE | $PS_1$ | $PS_2$ | $Pd_1$ | $Pd_2$ |
| PRÉ | Number of cases | 20 | 20 | 19 | 20 | 20 | 20 | 20 | 18 | 16 | 19 | 19 | 18 | 20 | 20 | 20 | 20 | 20 |
| | Values mx | 1.4 | 4.0 | 2.0 | 2.5 | 1.5 | 3.0 | 1.2 | 3.5 | 3.5 | 1.5 | 1.0 | 1.5 | 17.4 | 53.5 | 45.0 | 1.0 | 2.5 |
| | mn | -1.5 | -2.0 | -1.5 | -0.5 | -3.0 | -3.0 | -2.0 | -1.0 | -1.5 | -1.5 | -3.0 | -2.5 | 3.9 | 18.5 | 15.0 | -6.0 | -6.0 |
| | Arithmetic mean | 0.23 | 1.15 | 0.68 | 1.25 | -0.85 | -0.20 | -0.29 | 0.94 | 0.44 | -0.10 | -0.95 | -0.44 | 8.61 | 34.52 | 26.57 | -1.87 | -1.00 |
| | Standard deviation | 0.811 | 1.424 | 0.988 | 0.966 | 1.101 | 1.568 | 0.743 | 1.211 | 1.237 | 0.774 | 0.956 | 0.953 | 3.962 | 11.003 | 8.524 | 2.120 | 1.926 |
| PER | Number of cases | 19 | 19 | 19 | 19 | 18 | 19 | 18 | 18 | 17 | 18 | 17 | 17 | 18 | 18 | 18 | 18 | 18 |
| | Values mx | 2.6 | 2.5 | 3.5 | 3.0 | 2.0 | 2.5 | 2.0 | 5.0 | 3.5 | 2.0 | 0.5 | 2.5 | 18.6 | 64.0 | 55.0 | 2.5 | 2.0 |
| | mn | -0.5 | -0.5 | 0.0 | -0.5 | -1.5 | -2.0 | -1.2 | -1.0 | -1.0 | -1.0 | -3.0 | -1.5 | 5.8 | 22.0 | 16.0 | -5.0 | -2.0 |
| | Arithmetic mean | 0.40 | 0.87 | 1.13 | 1.13 | -0.36 | -0.29 | -0.09 | 0.69 | 0.68 | 0.44 | -1.06 | -0.38 | 11.76 | 41.05 | 33.47 | -0.72 | 0.11 |
| | Standard deviation | 0.751 | 0.779 | 0.796 | 0.895 | 0.801 | 1.170 | 0.721 | 1.351 | 1.131 | 0.662 | 1.072 | 0.976 | 3.821 | 11.955 | 11.612 | 2.102 | 1.231 |
| FECH | Number of cases | 15 | 15 | 14 | 15 | 15 | 15 | 16 | 15 | 15 | 16 | 15 | 15 | 15 | 15 | 15 | 15 | 15 |
| | Values mx | 8.7 | 15.0 | 8.0 | 9.5 | 7.0 | 9.0 | 1.0 | 5.5 | 5.0 | 1.5 | 0.5 | 2.0 | 16.3 | 55.0 | 46.0 | 2.5 | 2.5 |
| | mn | -0.3 | -0.5 | 0.0 | 0.0 | -6.5 | -1.5 | -1.0 | -0.5 | -0.5 | 0.0 | -2.5 | -1.5 | 5.6 | 10.0 | 16.0 | -3.5 | -2.0 |
| | Arithmetic mean | 2.52 | 3.67 | 3.36 | 3.37 | 0.87 | 2.00 | 0.19 | 1.00 | 1.17 | 0.75 | -0.80 | -0.07 | 8.56 | 28.77 | 27.63 | -0.23 | 0.27 |
| | Standard deviation | 2.787 | 4.443 | 2.560 | 2.545 | 3.512 | 3.185 | 0.558 | 1.669 | 1.305 | 0.632 | 0.751 | 0.883 | 2.970 | 12.159 | 8.123 | 1.699 | 1.294 |

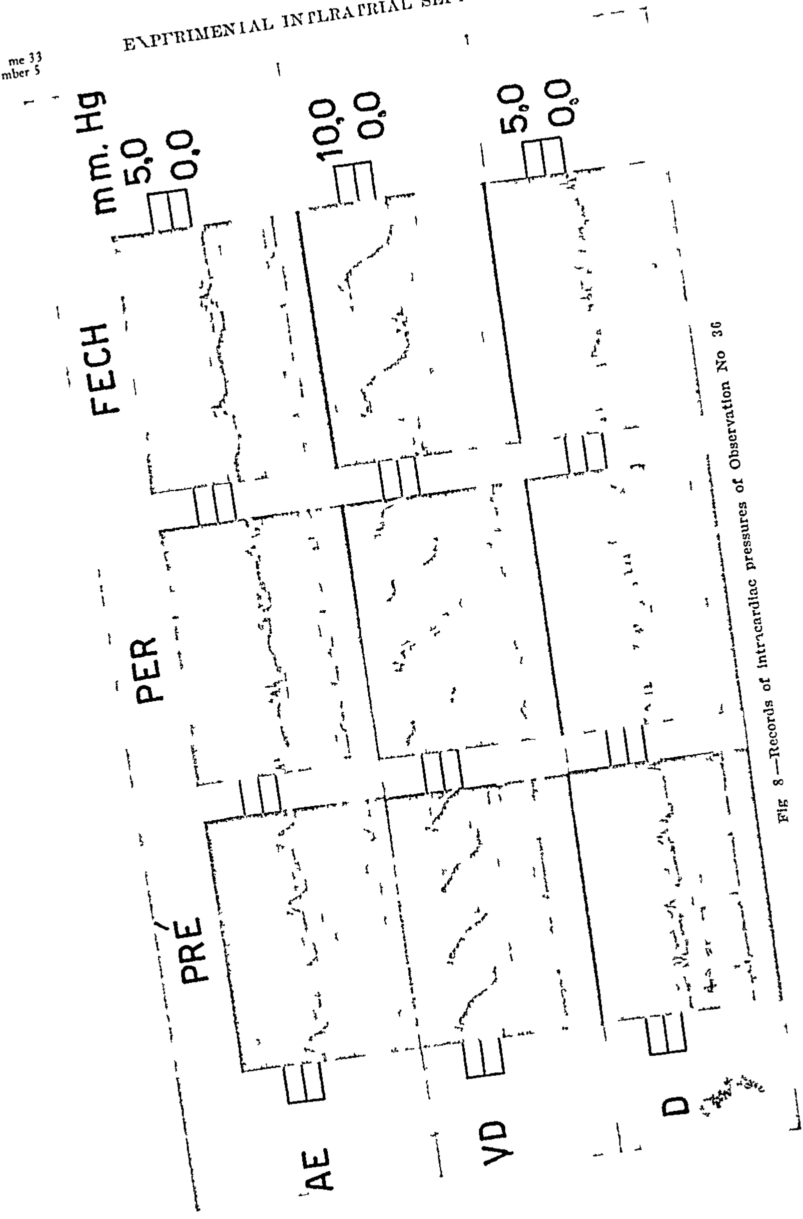


Fig 8—Records of intracardiac pressures of Observation No 36

TABLE II. SYNTHESIS OF THE RESULTS OF INTRACARDIAC PRESSURES OF CASES UNDER SINUSAL RHYTHM
(Animals operated on under artificial hibernation, mm. Hg)

| | | AE | | | | | | AD | | | | | | VD | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | MEAN PRES-SURE | A | B | D | A′ | Y | MEAN PRES-SURE | A | B | D | A′ | Y | MEAN PRES-SURE | $PS_1$ | $PS_2$ | $Pd_1$ | $Pd_2$ |
| PRÉ | Number of cases | 28 | 28 | 28 | 28 | 28 | 28 | 29 | 29 | 27 | 29 | 27 | 28 | 29 | 26 | 27 | 27 | 29 |
| | Values mx | 4.5 | 10.5 | 8.5 | 5.0 | 6.5 | 5.5 | 2.9 | 8.5 | 8.5 | 3.5 | 2.5 | 3.5 | 9.6 | 27.0 | 26.0 | 2.0 | 2.0 |
| | Values mn | -1.2 | -1.5 | 0.0 | -1.0 | -2.0 | -2.5 | -1.9 | -1.5 | -1.0 | -2.0 | -3.5 | -2.5 | 3.0 | 8.0 | 7.0 | -2.0 | -2.0 |
| | Arithmetic mean | 1.04 | 3.30 | 3.25 | 1.39 | 0.96 | 0.82 | -0.02 | 1.59 | 1.18 | -0.34 | -0.55 | -0.39 | 5.01 | 14.71 | 14.05 | -0.52 | -0.45 |
| | Standard deviation | 1.392 | 2.286 | 2.401 | 1.383 | 2.211 | 1.743 | 1.135 | 2.036 | 2.010 | 1.302 | 4.479 | 1.343 | 1.658 | 4.384 | 5.148 | 1.051 | 1.012 |
| PER | Number of cases | 27 | 25 | 27 | 27 | 25 | 25 | 24 | 24 | 24 | 24 | 23 | 24 | 24 | 23 | 25 | 22 | 24 |
| | Values mx | 3.1 | 6.0 | 6.5 | 4.0 | 4.0 | 3.0 | 2.8 | 3.5 | 5.0 | 3.0 | 2.5 | 2.5 | 15.1 | 42.5 | 37.5 | 1.0 | 2.5 |
| | Values mn | -1.5 | -1.0 | -1.0 | -1.0 | -2.5 | -3.0 | -1.4 | -2.0 | -1.5 | -1.0 | -2.5 | -2.0 | 2.3 | 10.0 | 9.0 | -4.0 | -3.5 |
| | Arithmetic mean | 0.41 | 1.38 | 1.87 | 0.91 | 0.18 | -0.10 | 0.10 | 1.12 | 1.60 | 0.52 | -0.33 | -0.35 | 6.94 | 18.69 | 17.88 | 0.27 | 0.02 |
| | Standard deviation | 1.040 | 1.657 | 1.790 | 1.225 | 1.523 | 1.283 | 2.220 | 1.279 | 1.525 | 1.047 | 1.230 | 0.983 | 2.761 | 8.563 | 6.850 | 1.120 | 1.229 |
| FECH | Number of cases | 12 | 12 | 12 | 12 | 12 | 12 | 9 | 8 | 8 | 9 | 6 | 7 | 10 | 10 | 11 | 10 | 11 |
| | Values mx | 2.0 | 3.0 | 5.0 | 4.0 | 2.0 | 2.0 | 1.4 | 3.5 | 5.0 | 1.5 | 1.5 | 1.0 | 9.5 | 26.0 | 21.5 | 2.0 | 2.5 |
| | Values mn | -0.3 | 0.0 | 1.5 | 0.0 | 0.0 | -1.0 | -1.5 | -1.0 | -1.5 | -1.5 | -2.0 | -1.5 | 3.9 | 10.0 | 8.5 | -1.0 | -2.0 |
| | Arithmetic mean | 0.75 | 1.54 | 3.00 | 1.71 | 0.67 | 0.21 | -0.11 | 0.87 | 1.06 | 0.11 | -0.83 | -0.43 | 5.63 | 16.05 | 16.23 | 0.15 | -0.45 |
| | Standard deviation | 0.762 | 1.033 | 1.348 | 1.322 | 0.807 | 0.916 | 0.931 | 1.529 | 2.078 | 1.083 | 1.505 | 1.159 | 3.339 | 5.036 | 4.502 | 1.180 | 1.213 |

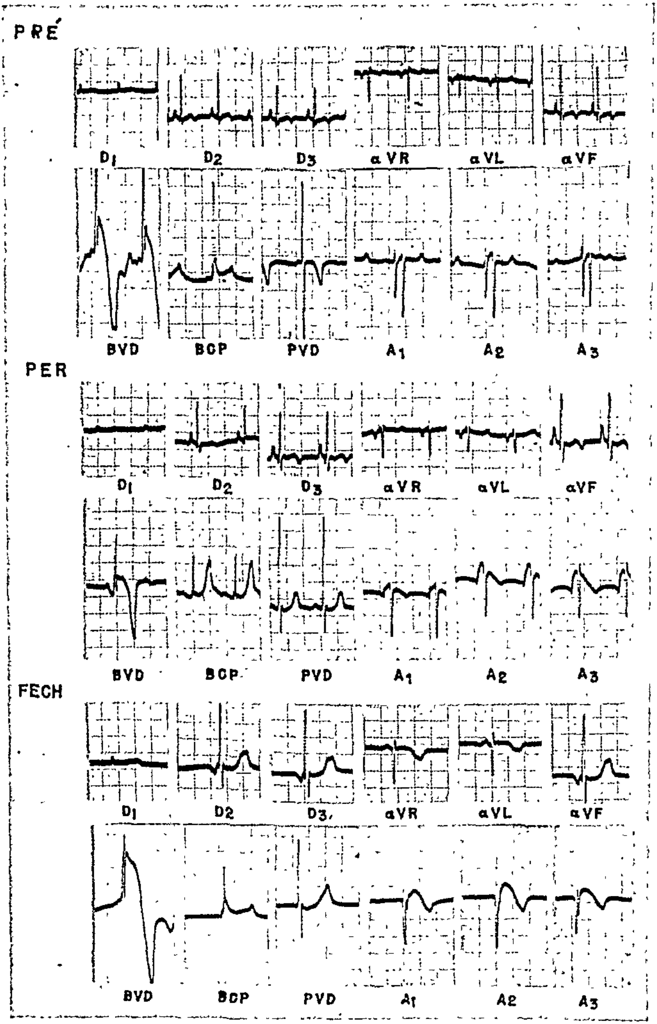


Fig. 9.—Electrocardiographic records of Observation No. 20.

TABLE III. SYNTHESIS OF THE RESULTS OF ELECTROCARDIOGRAPHIC RECORDS OF CLASSIC AND UNIPOLAR LEADS OF THE CASES UNDER SINUSAL RHYTHM (Animals operated on at normal temperature)

| | | WAVE P | | | | INTERVAL P-R | | COMPLEX QRS | | | | INTERVAL | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | DURATION (SEC.) | Pc | AMPLITUDE (MM.) | ÂP (DEGREES) | P-R (SEC.) | P-Rc | DURATION (SEC.) | QRSc | AMPLITUDE (MM.) | ÂQRS (DEGREES) | Q-T (SEC.) | Q-Tc |
| PRE | Number of cases | 20 | 20 | 20 | 20 | 20 | 20 | 20 | 20 | 20 | 19 | 19 | 20 |
| | Values { mx<br>mn | 0.055<br>0.030 | 0.061<br>0.033 | 8.5<br>2.5 | 90<br>65 | 0.110<br>0.060 | 0.119<br>0.066 | 0.060<br>0.030 | 0.097<br>0.047 | 57<br>23 | 95<br>20 | 0.340<br>0.166 | 0.447<br>0.250 |
| | Arithmetic mean | 0.0392 | 0.0442 | 5.06 | 84.3 | 0.0807 | 0.0906 | 0.0465 | 0.0747 | 37.4 | 76.1 | 0.2158 | 0.3507 |
| | Standard deviation | 0.008 | 0.009 | 1.584 | 6.961 | 0.013 | 0.014 | 0.008 | 0.013 | 9.439 | 20.230 | 0.029 | 0.039 |
| PER | Number of cases | 18 | 18 | 18 | 18 | 18 | 18 | 18 | 18 | 18 | 16 | 18 | 18 |
| | Values { mx<br>mn | 0.065<br>0.035 | 0.068<br>0.037 | 6.5<br>2.3 | 95<br>80 | 0.105<br>0.055 | 0.112<br>0.061 | 0.060<br>0.035 | 0.102<br>0.051 | 49<br>12 | 110<br>30 | 0.382<br>0.196 | 0.479<br>0.326 |
| | Arithmetic mean | 0.0441 | 0.0478 | 4.72 | 89.8 | 0.0861 | 0.0942 | 0.0508 | 0.0731 | 28.5 | 77.8 | 0.2762 | 0.4044 |
| | Standard deviation | 0.009 | 0.007 | 1.304 | 3.053 | 0.014 | 0.014 | 0.007 | 0.015 | 9.313 | 21.906 | 0.041 | 0.042 |
| FECH | Number of cases | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 15 | 13 | 13 | 14 |
| | Values { mx<br>mn | 0.055<br>0.030 | 0.062<br>0.032 | 8.0<br>2.5 | 115<br>70 | 0.110<br>0.050 | 0.126<br>0.057 | 0.060<br>0.040 | 0.100<br>0.063 | 51<br>19 | 100<br>20 | 0.316<br>0.214 | 0.437<br>0.290 |
| | Arithmetic mean | 0.0407 | 0.0448 | 4.84 | 90.5 | 0.0800 | 0.0891 | 0.0520 | 0.0779 | 30.7 | 83.5 | 0.2653 | 0.3837 |
| | Standard deviation | 0.007 | 0.008 | 1.391 | 10.020 | 0.016 | 0.018 | 0.007 | 0.012 | 10.688 | 20.955 | 0.032 | 0.036 |

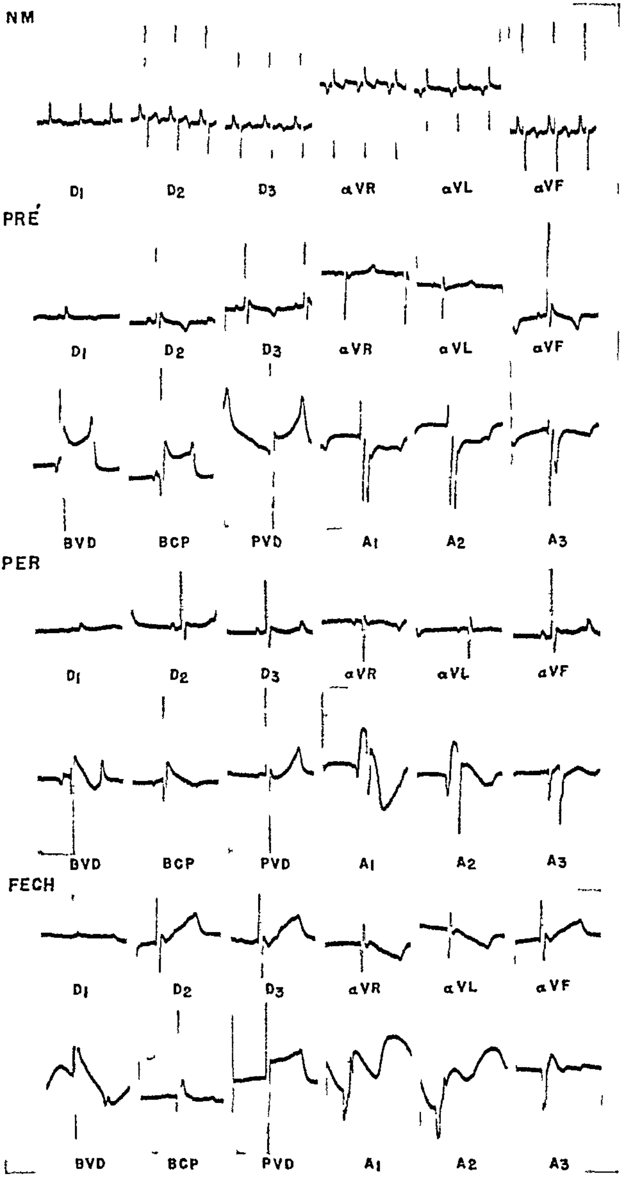


Fig 10—Electrocardiographic records of Observation No 41

TABLE IV. SYNTHESIS OF THE RESULTS OF ELEC[illegible]RDIOGRAPHIC RECORDS OF CLASSIC AND UNIPOLAR LEADS OF THE CASES UNDER SINUSAL RHYTHM (Hibernated animals)

| | | WAVE P | | | | INTERVAL P-R | | COMPLEX QRS | | | | INTERVAL | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | DURATION (SEC.) | Pc | AMPLITUDE (MM.) | ÂP (DEGREFS) | P-R (SEC.) | P-Rc | DURATION (SEC.) | QRSc | AMPLITUDE (MM.) | ÂQRS (DEGREES) | Q-T (SEC.) | Q-Tc |
| NM | Number of cases | 10 | 10 | 9 | 9 | 10 | 10 | 10 | 10 | 10 | 10 | 10 | 10 |
| | Values mx | 0.040 | 0.046 | 7.3 | 90 | 0.110 | 0.118 | 0.045 | 0.085 | 95 | 110 | 0.308 | 0.545 |
| | mn | 0.030 | 0.035 | 3.3 | 35 | 0.060 | 0.069 | 0.030 | 0.050 | 38 | 60 | 0.168 | 0.283 |
| | Arithmetic mean | 0.0365 | 0.0410 | 5.40 | 77.2 | 0.0770 | 0.0874 | 0.0365 | 0.0638 | 55.5 | 84.0 | 0.2028 | 0.3504 |
| | Standard deviation | 0.005 | 0.008 | 1.471 | 16.895 | 0.014 | 0.014 | 0.006 | 0.010 | 18.477 | 13.292 | 0.042 | 0.041 |
| PRE | Number of cases | 10 | 10 | 10 | 10 | 10 | 10 | 10 | 10 | 10 | 10 | 9 | 9 |
| | Values mx | 0.070 | 0.073 | 10.0 | 115 | 0.130 | 0.138 | 0.080 | 0.130 | 92 | 135 | 0.658 | 0.639 |
| | mn | 0.040 | 0.048 | 1.0 | -30 | 0.090 | 0.104 | 0.055 | 0.058 | 29 | 65 | 0.350 | 0.385 |
| | Arithmetic mean | 0.0555 | 0.0580 | 4.09 | 69.0 | 0.1145 | 0.1172 | 0.0640 | 0.0783 | 47.9 | 89.7 | 0.4184 | 0.4761 |
| | Standard deviation | 0.010 | 0.015 | 2.679 | 41.352 | 0.016 | 0.014 | 0.008 | 0.020 | 19.011 | 18.583 | 0.104 | 0.081 |
| PER | Number of cases | 7 | 7 | 7 | 7 | 7 | 7 | 7 | 7 | 7 | 7 | 7 | 7 |
| | Values mx | 0.105 | 0.106 | 7.5 | 90 | 0.320 | 0.323 | 0.100 | 0.108 | 47 | 115 | 0.786 | 0.664 |
| | mn | 0.045 | 0.052 | 2.2 | 80 | 0.095 | 0.099 | 0.055 | 0.055 | 19 | 85 | 0.423 | 0.449 |
| | Arithmetic mean | 0.0650 | 0.0657 | 4.23 | 87.4 | 0.1592 | 0.1604 | 0.0707 | 0.0747 | 31.8 | 95.7 | 0.5064 | 0.5302 |
| | Standard deviation | 0.021 | 0.019 | 2.155 | 3.823 | 0.076 | 0.076 | 0.017 | 0.018 | 10.930 | 10.177 | 0.127 | 0.070 |
| FECH | Number of cases | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 |
| | Values mx | 0.080 | 0.079 | 5.0 | 120 | 0.220 | 0.209 | 0.110 | 0.124 | 47 | 105 | 0.652 | 0.600 |
| | mn | 0.040 | 0.041 | 1.5 | 80 | 0.105 | 0.108 | 0.080 | 0.087 | 16 | -60 | 0.434 | 0.472 |
| | Arithmetic mean | 0.0700 | 0.0687 | 3.35 | 95.0 | 0.1600 | 0.1555 | 0.0950 | 0.0912 | 27.5 | 58.7 | 0.5565 | 0.5265 |
| | Standard deviation | — | — | — | — | — | — | — | — | — | — | — | — |
| AQ | Number of cases | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 |
| | Values mx | 0.040 | 0.045 | 10.0 | 90 | 0.090 | 0.124 | 0.050 | 0.079 | 58 | 120 | 0.278 | 0.452 |
| | mn | 0.030 | 0.033 | 3.2 | 85 | 0.063 | 0.070 | 0.035 | 0.042 | 25 | 85 | 0.178 | 0.255 |
| | Arithmetic mean | 0.0340 | 0.0386 | 5.84 | 88.4 | 0.0796 | 0.0952 | 0.0390 | 0.0626 | 37.8 | 96.2 | 0.2122 | 0.3668 |
| | Standard deviation | 0.004 | 0.015 | 2.748 | 2.213 | 0.015 | 0.022 | 0.007 | 0.017 | 15.172 | 14.567 | 0.042 | 0.032 |

3 Artificial hibernation, still in the developmental phase, offered favorable conditions for the performance of surgical intracardiac maneuvers

4 Artificial hibernation induced hemodynamic changes, evidenced by variations in the pressure values and the morphology of pressure curves

5 There was significant predominance of the mean pressure interatrial gradient from left to right in all the experimental stages in dogs operated on at normal temperature

6 There was significant predominance from left to right of the interatrial gradient of the B, A′, and Y waves after closure of the IASD, in the series of dogs operated on at normal temperature

7 The interatrial gradient of the mean and instantaneous pressures showed a significant predominance from left to right in dogs with open chest and intact interatrial septum in the series submitted to artificial hibernation

8 Significant predominance from left to right of the D waves interatrial pressure gradient observed in all the experimental stages in both series of animals

9 Significant changes of the atrial pressures observed in the series of dogs operated on at normal temperature, after creation of the IASD, were (a) pressure increase of the D wave in the right atrium, and (b) decrease of the D wave interatrial pressure gradient

10 Significant changes of the atrial pressures observed in the series operated on under artificial hibernation, after creation of the IASD, were (a) decrease of mean and instantaneous pressures in the left atrium, (b) increase of the wave pressure in the right atrium, and (c) decrease of the interatrial gradient of the mean and D wave pressures

11 Significant changes of the atrial pressures observed in the series of dogs operated on at normal temperature, after closure of the IASD, were (a) increase of mean and instantaneous pressures in the left atrium, and (b) increase of the interatrial pressure gradient of the D wave

12 There was a significant increase of the D wave pressure in the left atrium, in the series of dogs operated on under artificial hibernation, after closure of the IASD

13 A comparison of the values observed after closure of the interatrial septal defect with those before its production, in dogs operated on at normal temperature, showed (a) significant increase of the mean and instantaneous pressures in the left atrium, and (b) significant increase of the instantaneous pressures B, D, and Y in the right atrium

14 Values of the mean and instantaneous pressures A, B, D, and Y observed after closure of the interatrial septal defect, in the series of animals operated on under artificial hibernation, were, in the left atrium, significantly lower than those registered before its creation

15 No significant changes of the mean values in the right atrium were observed after production and closure of the IASD in both series of animals

16 Mean and systolic pressures in the right ventricle, before the creation of the interatrial septal defect in animals operated upon under artificial

In our experiments, closure of the defect was made by two different methods in the two groups of animals. In one of them, we used the method of Gross (1952), in the other, closure under direct vision. In both groups, the defect was completely closed with three stitches.

Coronary air embolism, a frequent cause of death early in this series, was prevented by placing a Satinsky clamp in such a way as to occlude the outflow of the heart through the aorta and pulmonary artery, and flooding the cardiac and thoracic cavities with saline solution.

Few studies are found in the literature on the changes of intracardiac pressures determined by the creation and closure of the experimental IASD and practically none under hibernation. The study of the instantaneous pressures in the several experimental phases was made by pressure tracings recorded simultaneously with the electrocardiogram.

Some features of the changes induced by experimentation in the right ventricle were observed by the pressure tracings in this cavity.

The correlation test between the average pressure and the frequency demonstrated that the pressure increase of the right ventricle, after production of the defect, was due, not to the frequency but, most probably, to the blood flow increase in this cavity.

The phenomenon explained by Sarnoff and Berglund (1954) as the shift of the heart from one "ventricular function curve" at a certain level to another at a higher level, in which with less filling pressure a greater output is obtained, was observed in one-third of the cases.

The presence of the "form of resistance" of Borges, characterized by the predominance of $Ps_2$ over $Ps_1$, was observed.

Electrocardiographic records rendered possible the observation of some electrocardiographic changes of the heart in the several experimental phases. It seems likely that the electrocardiographic changes observed are fundamentally due to the general conditions peculiar to the experiment and not to the hemodynamic changes determined by the defect.

The electrocardiographic changes caused by hibernation were more uniform and their findings are similar to those reported by other authors.

Admitting the existence of a relationship between the electrocardiographic and mechanical phenomena, an attempt was made to correlate the hemodynamic changes observed to the electrocardiographic ones. No Pearson coefficient, statistically significant at the level of one and 5 per cent, was found among the several elements studied.

## CONCLUSIONS

1. The creation of a large interatrial septal defect under direct vision was well tolerated by the dog.

2. Clamping of the aorta up to the origins of the coronary arteries and flooding of the cardiac and chest cavities with saline solution were adequate measures to prevent coronary air embolism in the closure of IASD under direct vision.

of the P R interval, (d) a tendency toward increase in duration of the QRS complex, and (e) a tendency toward increase of Q-T values

29 In the series of animals operated on under artificial hibernation, there was reversibility of electrocardiographic changes due to the tendency to return, after rewarming, to the values observed before hibernation

## REFERENCES

1 Adams, W E, and Escudero, L Disturbances in the Circulation and Respiration in Obstruction of Blood Flow To and From the Heart, Surg, Gynec & Obst 70 744 752, 1940

2 Albers, D, and Bedbuhr, W 1941, Cit by Lepeschkin, E, 1951, p 171, Arch Kreislaufforsch 8 150, 1941

3 Bahnson, H T, and Bacher, C B Study of Prosthetic Materials Used Inside the Heart, Surg Forum, 78 83, 1953

4 Bailey, C P, Cookson, B A, Downing, D F, and Neptune, W B Cardiac Surgery Under Hypothermia, J THORACIC SURG 27 73 95, 1954

5 Bairão, G B Hibernação Artificial, II Congr Lat Amer Anestesiol e I Congr Brasil Anestesiol, São Paulo, 1954

6 Barbato, E C D Estudo eletrocardiográfico da ativação ventricular normal e patológica. Sua importancia na interpretação das chamadas curvas de hipertrofia, Tese inaug Fac Med Univ S Paulo, 1952

7 Bigelow, W G, Callaghan, J C, and Hopps, J A General Hypothermia for Experimental Intracardiac Surgery The Use of Electrophrenic Respirations, an Artificial Pacemaker for Cardiac Standstill, and Radio Frequency Rewarming in General Hypothermia, Ann Surg 132 531 539, 1950 a

8 Bigelow, W G, Lindsay, W K, and Greenwood, W F Hypothermia Its Possible Role in Cardiac Surgery An Investigation of Factors Governing Survival in Dogs at Low Body Temperatures, Ann Surg 132 849 866, 1950 b

9 Bjork V O, and Crafoord, C The Surgical Closure of Interauricular Septal Defects, J THORACIC SURG 26 300 308, 1953

10 Blalock, A, and Hanlon, C R Interatrial Septal Defect Its Experimental Production Under Direct Vision Without Interruption of the Circulation, Surg, Gynec & Obst 87 183 187, 1948

11 Boerema, I, Wildschut, A, Schmidt, W J H, and Broekhuysen, L Experimental Researches Into Hypothermia as An Aid in Surgery of the Heart, Arch chir neerl 3 25 34, 1951

12 Borges, S, Ramos, O, Nicolai, A, San Juan, E, Zerbini, E J, Portugal, O, Losso, L, and Nunes, A Aspectos hemodinamicos observados em 8 casos de comissurotomia mitral Estudo pre e pós operatório, Arch brasil cardiol 6 157 186, 1953

13 Borges S Campos Filho, C M, Carvalhal, S, Losso, L, Zerbini, E J, Buzzi, R, and Viana F C Estudo das pressões na auricula esquerda durante o tratamento cirurgico de 26 casos de molestia mitral Arch brasil cardiol 7 175 190, 1954

14 Borges, S Comunicação pessoal, 1955

15 Borrie, J Closure of Experimental Atrial Septal Defects in Sheep With a Special Atrial Ring Clamp, J THORACIC SURG 29. 426 434, 1955

16 Botwin, A E Pedunculated Atrial Flaps Used for Experimental Cardiac Surgery, J THORACIC SURG 28 300 301, 1954

17 Brecher, G A., and Opdyke, D F Effect of Normal and Abnormal Respiration on Hemodynamics of Experimental Interatrial Septal Defects, Am J Physiol 162 507 520, 1950

18 Brecher, G A, and Opdyke, D F The Relief of Acute Right Ventricular Strain by the Production of an Interatrial Septal Defect, Circulation 4 496 502, 1951

19 Cahn, J, Melon, J M, and Dubrasquet, M Le coeur sous hibernation artificielle, Anesth et analg 10 420 425, 1953 a.

20 Cahn, J, Dubrasquet, M, Bodiou, J, and Melon, J M Methodes d'hibernation artificielle Étude expérimentale comparée, Therapie 8 808 901, 1953 b

21 Cahn, J, Melon, J M., and Dubrasquet, M Chirurgie cardiaque expérimentale et hibernation artificielle, Therapie 8 812 817, 1953 c

22 Cheer, S N The Effects of High Temperature on Heart and Circulation of Intact Animals, Am J Physiol 84 587 598, 1928

23 Chiaverini, R Contribuição para o estudo eletrocardiografico da sobrecarga ventricular direita, Tese, Fac Med Univ S Paulo, Imp Ipsis, 110, 1950

24 Cohen, M, Hammerstrom, R N, Spellman, M W, Varco, R L, and Lillehei, C W The Tolerance of the Canine Heart to Temporary Complete Vena Caval Occlusion, Surg Forum, 172 177, 1952

hibernation, were significantly lower than in those operated at normal temperature and $Pd_1$ value was significantly higher.

17. There was a significant increase of the mean pressures in the right ventricle, in both series of animals, after creation of the defect and significant decrease after its closure.

18. Systolic and $Pd_2$ pressures in the right ventricle were significantly increased after production of the IASD, in both series of animals.

19. Significant decrease of systolic pressures in the right ventricle was observed after closure of the defect in the dogs operated on at normal temperature.

20. Significant decrease of cardiac frequency after closure of the IASD was observed in the series of animals operated on at normal temperature.

21. Progressive and significant decrease of cardiac frequency in the various experimental stages was observed in the series of animals operated upon under artificial hibernation.

22. The incidence of supraventricular rhythms of the nonsinusal type was higher after closure of the defect, mainly in animals operated upon under artificial hibernation.

23. Main electrocardiographic changes observed after the production of the IASD, in the series of animals operated upon at normal temperature, were: (a) a tendency toward increase of the P-wave duration; (b) a significant deviation of P-angle to the right; (c) a tendency toward increase of the P-R interval; (d) a tendency toward increase of the Q-T interval; and (e) a significant shortening of the R/S relation obtained by direct epicardial lead and recorded at the base of the right ventricle.

24. Main electrocardiographic changes after creation of the IASD, in animals operated on under artificial hibernation, were: (a) tendency toward increase of the P-R interval; and (b) tendency toward increase of the Q-T interval.

25. No uniform or significant electrocardiographic changes were observed after closure of the IASD in the series of animals operated upon at normal temperature.

26. The comparison of electrocardiographic values observed after closure of the defect to those registered before its production, in the series of dogs operated upon at normal temperature, showed: (a) a tendency toward increase of the Q-T interval; and (b) a significant decrease of the R/S relation recorded at the base of the right ventricle.

27. The morphologic changes in the RS-T segment and T wave observed in animals operated on under artificial hibernation were different from those of animals operated upon at normal temperature and ones usually found in cardiac surgery.

28. The comparison of values registered in dogs at normal temperature to those observed in the same animals after hibernation and with open chest showed: (a) a tendency toward increase in duration of the P waves; (b) a significant decrease of the P-wave amplitude; (c) a tendency toward increase

53 Lam, C R Cit by Bailey, C P, Cookson, B A, Downing, D F, and Neptune, W B, 1954

54 Lange, K, Weiner, D, and Gold, M M Studies on the Mechanism of Cardiac Injury in Experimental Hypothermia, Ann Int Med 31 898 1002, 1949

55 Langendorf, F O 1895—Cit by Hegnauer, A H, Shriber, W J, and Haterius, H O, 1950

56 Laufman, H Profound Accidental Hypothermia, J A M A 147 1201 1212, 1951

57 Leeds, S L Cit by Swan, H, Maresh, G, Johnson, M E and Warner, G, 1950

58 Lepeschkin, E Modern Electrocardiography The P Q R S T U Complex, Baltimore, 1951, Williams & Wilkins Co, vol 1

59 Lepeschkin, E Comunicação pessoal, 1955

60 Lewis F J, and Taufic, M Closure of Atrial Septal Defects With the Aid of Hypothermia, Experimental Accomplishments and the Report of One Successful Case, Surgery 33 52 59, 1953

61 Lillehei, C W, Cohen, M, Spellmann, M W, and Hammerstrom, R N The Effect of Interatrial Septal Defect Shunts Upon Susceptibility to Bacterial Endocarditis Surg Forum 51 59, 1953

62 Little, R C, Opdyke, D F, and Hawley, J G Dynamics of Experimental Atrial Septal Defects, Am J Physiol 158 241 250, 1949

63 Marion, P, and Termet, H Traitement chirurgical de la communication inter auriculaire Travail experimental, Lyon chir 45 583 588, 1950

64 Martin, W B, and Essex, H E Production and Closure of Atrial Septal Defects in the Dog Observations on Atrial Pressures, Am J Physiol 155 453, 1948

65 Martin, W B, and Essex, H E Experimental Production and Closure of Atrial Septal Defects With Observations of Physiologic Effects, Surgery 30 283 297, 1951

66 McMichael, J Dynamics of Heart Failure, Brit M J 2 525 529, 1952

67 Miller, B J, Gibbon, J H, Jr, Greco, V F, Smith, B A, Cohn, C H, and Albritten, F F, Jr The Production and Repair of Interatrial Septal Defects Under Direct Vision With the Assistance of an Extracorporeal Pump Oxygenator Circuit, J Thoracic Surg 26 598 616, 1953

68 Moore, T C, and Shumacker, H B Jr Experimental Creation of Atrial Septal Defects, With Some Notes on the Production of a Right to Left Atrial Shunt, Angiology 6 244 252, 1953

69 Murray, G Closure of Defects in Cardiac Septa, Ann Surg 128 843 853, 1948

70 Mustard, W T Chute, A L, and Simmons, E H Further Observations on Experimental Extracorporeal Circulation Surgery 32 803 810, 1952

71 Opdyke, D F, Duomarco, J, Dillon, W H, Schreiber, H, Little, R C, and Seely, R D Study of Simultaneous Right and Left Atrial Pressure Pulses Under Normal and Experimentally Altered Conditions, Am J Physiol 154 258 272, 1948

72 Opdyke, D F, and Brecher, G A Modifying Effects of Interatrial Septal Defect on Cardiodynamics of Mitral Stenosis, Am J Physiol 164 573 582, 1951

73 Prec, O, Roenman, R, Braun, K, Rodbard, S and Katz, L N The Cardiovascular Effects of Acutely Induced Hypothermia J Clin Invest 28 293 300, 1949

74 Remington, J W, and Huggins R A Relation of Left Ventricular Ejection Period to the QT Interval of the Electrocardiogram Am J Physiol 175 185 190 1953

75 Ripstein, C B, Friedgood, C E, and Solomon, N A Technique For the Production of Hypothermia, Surgery 35 98 103, 1954

76 Sarnoff, S J and Berglund, E Ventricular Function I Starling's Law of the Heart Studied by Means of Simultaneous Right and Left Ventricular Function Curves in Dog, Circulation 9 706 718 1954

77 Senning, A Ventricular Fibrillation During Extracorporeal Circulation Used as a Method to Prevent Air Embolism and to Facilitate Intracardiac Operations, Acta chir scandinav, supp 171, 179, 1952

78 Shumacker, H B, Jr Surgical Repair of Atrial Septal Defects, Ann Surg 138 404 414, 1953

79 Shumacker, H B, Jr, and Moore, T C Surgical Closure of Atrial Septal Defects A Preliminary Report, Quart Bull Indiana Univ M Center 15 3 4, 1953

80 Shumacker, H B, Jr Moore, T C, and King, H The Experimental Closure of Atrial Septal Defects, J Thoracic Surg 26 551 573, 1953

81 Shumacker, H B, Jr, King, H and Lurie, P R Further Observations on the Closure of Atrial Septal Defects, Circulation 9 504 510, 1954

82 Sodi Pallares, D Nuevas bases de la electrocardiografia Instituto Nacional de Cardiologia, ed 3, Prensa medica Mexicana, Mexico, 1951

83 Sodi Pallares, D 1955 Comunicaçao pessoal

84 Sondergaard, T 1952, Cit by Bjork, V O, and Crafoord, C, 1953

85 Sondergaard, T, Sorensen, H R, Poulsen, T, and Andersen, I Experimental Production of Atrial Defects Under Direct Vision, Acta chir scandinav 107 477-484, 1954 a

25. Cohn, R.: The Experimental Method For the Closure of Interauricular Septal Defects in Dogs, Am. Heart J. 33: 453-457, 1947.

26. Cookson, B. A., Neptune, W., and Bailey, C. P.: Intracardiac Surgery With Hypothermia, J. Internat. Coll. Surgeons 18: 685-694, 1952 a.

27. Cookson, B. A., Neptune, W., and Bailey, C. P.: Hypothermia as a Means of Performing Intracardiac Surgery Under Direct Vision, Dis. Chest 22: 245-260, 1952 b.

28. Cookson, B. A., Neptune, W., and Bailey, C. P.: Intracardiac Surgery as an Adjunct to Hypothermia in the Repair of Septal Defects, Ann. Surg. 140: 100-106, 1954.

29. Décourt, L. V.: A sistole elétrica ventricular. Métodos de apreciação, valores normais e desvios em estados patológicos, Tese, Fac. Med. Univ. S. Paulo, Melhoramentos, 178 pp., 1949.

30. Dennis, C., Spreng, D. S., Jr., Nelson, G. E., Karlson, K. E., Nelson, R. M., Thomas, J. V., Edder, W. P., and Varco, R. L.: Development of a Pump-Oxygenator to Replace the Heart and Lungs; an Apparatus Applicable to Human Patients, and Application to One Case, Ann. Surg. 134: 709-721, 1951.

31. Dmitrieff, I. P.: Experimentelle Beitraege zur Frage ueber den operativen Zugang zu den Herzklappen und der Vorhofsscheidewand, Zentralbl. f. chir. 53: 715-718, 1926.

32. Dodrill, F. D.: A Method for Exposure of the Cardiac Septa. An Experimental Study, J. THORACIC SURG. 18: 652-660, 1949.

33. Donald, D. E., Kirklin, J. W., and Grindlay, J. H.: The Use of Polyvinyl Sponge Plugs in the Closure of Large Atrial Defects Created Experimentally, Proc. Staff Meet., Mayo Clin. 28: 288-295, 1953.

34. Dubost, C., d'Allaines, C., Tuffier, J., Passelecq, J., Madier, C., and Latscha, I.: Les possibilités d'asséchement des cavités cardiaques par l'hexaméthonium et le clampage simultané des veines caves. Essais de chirurgie intracardiaque à vue directe, Mém. Acad. Chir. 78: 307-313, 1952.

35. Fauteux, M.: Présentation d'instruments, Rev. Cand. biol. 7: 293-295, 1948.

36. Fujiwara, T.: 1939, Cit. by Lepeschkin, E., 1951, p. 130.

37. Glenn, W. W. L., and Sewell, W. H., Jr.: Experimental Cardiac Surgery. IV. The Prevention of Air Embolism in Open Heart Surgery; Repair of Interauricular Septal Defects, Surgery 34: 195-206, 1953.

38. Glover, R. P., Henderson, A. R., Margutti, R., and Gregory, J.: The Fate of Intracardiac Pericardial Grafts as Applied to the Closure of Septal Defects and to the Relief of Mitral Insufficiency, Surg. Forum, 178-185, 1952.

Gross, R. E., Pomeranz, A. A., Watkins, E., Jr., and Goldsmith, E. I.: Surgical Closure of Defects of the Interauricular Septum by Use of an Atrial Well, New England J. Med. 247: 455-460, 1952.

Gross, R. E., Watkins, E., Jr., Pomeranz, A. A., and Goldsmith, E. I.: A Method for Surgical Closure of Interauricular Septal Defects, Surg., Gynec. & Obst. 96: 1-23, 1953.

Haecker, R.: Experimentelle Studien zur Pathologie und Chirurgie des Herzens, Arch. f. klin. Chir. 84: 1035, 1907.

Hamilton, J. B., Dresbach, M., and Hamilton, R. S.: Cardiac Changes During Progressive Hypothermia, Am. J. Physiol. 118: 71-76, 1937.

Hegnauer, A. H., Shriber, W. J., and Haterius, H. O.: Cardiovascular Response of the Dog to Immersion Hypothermia, Am. J. Physiol. 161: 455-465, 1950.

Hegnauer, A. H., Flynn, J., and d'Amato, H.: Cardiac Physiology in Dog During Rewarming From Deep Hypothermia, Am. J. Physiol. 167: 69-75, 1951.

Helmsworth, J. A., Clark, L. C., Jr., Kaplan, S., and Sherman, R. T.: An Oxygenator-Pump for Use in Total By-Pass of Heart and Lungs. Laboratory Evaluation and Clinical Use, J. THORACIC SURG. 26: 617-632, 1953.

Hook, W. E., and Stormont, R. T.: Effect of Lowered Body Temperature on Heart Rate, Blood Pressure and the Electrocardiogram, Am. J. Physiol. 133: 334, 1941.

Hufnagel, C. A., and Gillespie, J. F.: Closure of Interauricular Septal Defects, Bull. Georgetown Univ. Med. Center 4: 137-139, 1951.

Hull, E.: The Cause and Effects of Flow Through Defects of the Atrial Septum, Am. Heart J. 38: 350-360, 1949.

Juvenelle, A. A., Lind, J., and Wegelius, C.: A New Method of Extracorporeal Circulation. Deep Hypothermia Combined With Artificial Circulation, Am. Heart J. 47: 692-736, 1954.

Kiriluk, L. B., Hoag, W., and Merendino, K. A.: Experimental Interauricular Septal Defects: A Physiologic Study With Evaluation of Methods of Closure, Surg. Forum 199-204, 1951.

Klakeg, C. H., Berry, A. M., and Burchell, H. B.: Duration of QT Interval at Slow Ventricular Rates, Am. J. Physiol. 179: 159-164, 1954.

Laborit, H., and Huguenard, P.: Pratique de l'hibernothérapie en chirurgie et en médecine, Paris, 1954, Masson & Cie.

# BRONCHOSPASM IN BRONCHOGRAPHY

Thomas H Hewlett, Lieutenant Colonel, MC, USA,[*] Anthony J Puglisi, Major, USAF (MC),[**] and Warner F Bowers, Colonel, MC, USA[***]
Fort Sam Houston, Texas

A LARGE number of patients with chronic cough eventually have broncho graphic studies performed to determine the presence of bronchiectasis Adequate bronchograms offer the only absolute proof of bronchiectasis and serve as the basis for excisional surgery Although a single bronchogram reveals the anatomic state of the bronchi at the time of film exposure, it does not offer unequivocal proof of irreversible pathologic changes within the bronchi Those experienced in pulmonary diseases recognize that the bronchogram alone cannot be accepted as the absolute criterion of lung damage or of the need for excisional surgery The proof of pulmonary damage is reflected in the patient's clinical course

The interpretation of bronchograms and the decision for excisional surgery is often delegated to the younger men in a training program Consequently, the possibility of mistaken interpretation of a single bronchogram might result in unwarranted operation Several factors may produce variations in bronchial filling which render the recognition of bronchiectasis difficult in adequate bronchograms Among those that are well recognized, the most prominent are acute respiratory infections, emphysema, and atelectasis Bronchial changes, which have been ascribed to chronic bronchitis or asthma, consist primarily of some degree of mild dilatation of the bronchi with beading and irregularities of caliber noted on bronchogram Little attention has been directed toward the possibility of bronchospasm occurring during bronchography which suggests that it is rare The experience at Brooke Army Hospital demonstrates that bronchospasm may account for a bronchogram which is interpreted as showing bronchiectasis

Seventy nine bronchographic studies were performed at Brooke Army Hospital over an eighteen month period Bronchospasm during bronchography was encountered and proved in 7 patients of this group The significance of this 8 8 per cent incidence of bronchospasm during bronchography is of interest because the initial radiologic interpretation of these bronchograms was of bronchiectasis, usually bilateral These patients were not submitted to operation

---

From Thoracic Vascular Surgery Section Department of Surgery Brooke Army Hospital Fort Sam Houston Texas

Received for publication April 9 1956

[*] Chief Thoracic Vascular Surgery Section Assistant Chief General Surgery Service Assistant Professor of Surgery Graduate School Baylor University Houston Texas

[**] Formerly Surgical Resident at Brooke Army Hospital now assigned to USAF Hospital Clarke AFB Luzon P I

[***] Chief Department of Surgery and Chief General Surgery Service Brooke Army Hospital Fort Sam Houston Texas Chief Clinical Surgery Medical Field Service School Surgical Consultant 4th Army Headquarters Professor of Surgery Graduate School Baylor University Houston Texas

86. Sondergaard, T., Sorensen, H. R., Poulsen, T., and Andersen, I.: Closure of Experimentally Produced Atrial Septal Defects, Acta chir. Scandinav. 107: 485-491, 1954 b.
87. Stead, E. A., Jr., and Warren, J. V.: Cardiac Output in Man. An Analysis of the Mechanism Varying the Cardiac Output Based on Recent Clinical Studies, Arch. Int. Med. 80: 237-248, 1947.
88. Swan, H., Maresh, G., Johnson, M. E., and Warner, G.: The Experimental Creation and Closure of Auricular Septal Defects, J. THORACIC SURG. 20: 542-551, 1950.
89. Swan, H., and Stewart, B. D.: A Modified Button Technique for the Closure of Experimental Interauricular Septal Defects, J. THORACIC SURG. 25: 397-401, 1953.
90. Swan, H., Zeavin, I., Blount, S. G., Jr., and Virtue, R. W.: Surgery by Direct Vision in the Open Heart During Hypothermia, J. A. M. A. 153: 1081-1085, 1953.
91. Swan, H., and Zeavin, I.: Cessation of Circulation in General Hypothermia. III. Technics of Intracardiac Surgery Under Direct Vision, Ann. Surg. 139: 385-396, 1954.
92. Templeton, J. Y., III, and Gibbon, J. H., Jr.: Experimental Reconstruction of Cardiac Valves by Venous and Pericardial Grafts, Ann. Surg. 129: 161-176, 1949.
93. Turk, L. N., III, and Glenn, W. W. L.: Diverticulum Approach to the Heart Chambers: Repair of Defects in the Cardiac Septa by a Many-Tailed Plaque Technique, Surgery 37: 427-439, 1955.
94. Watkins, E., Jr., Pomeranz, A. A., and Gross, R. E.: Healing Patterns Following Closure of Experimental Defects of the Atrial Septum, Surg. Forum, 46-51, 1953.
95. Wenckebach and Winterberg: 1927, Cit. by van Bogaert, A., van Genabeek, A., Nyssens, A., van der Henst, A., and Vandael, J., 1952, Morphologie du pouls "capillaire" pulmonaire, Arch. mal. coeur 45: 673-698.
96. Zeavin, I., Virtue, W., and Swan, H.: Cessation of Circulation in General Hypothermia. II. Anesthetic Management, Anesthesiology 15: 113-121, 1954.

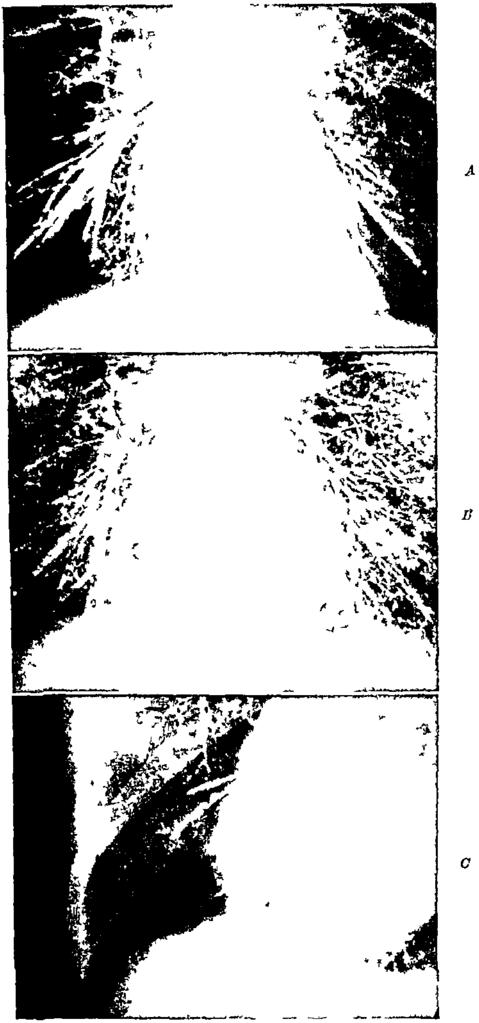


Fig 1 —*A* Initial posteroanterior bronchogram revealing bilateral bronchogram *B* Repeat posteroanterior bronchogram revealing normal patterns *C,* Left anterior oblique bronchogram revealing spasm right middle and lower lobes

because of the presentation of the bronchograms before a combined medico-surgical conference where experienced clinicians disagreed with the diagnosis of bronchiectasis and requested that the bronchographic studies be repeated. The repeat studies carried out on each patient resulted in essentially normal bronchograms. Thus, the value of conference interpretation of films is demonstrated as well as the fallacy of a single bronchographic study in patients whose clinical course, though suggestive of bronchiectasis, is not typical.

Bronchospasm is usually associated with the asthmatic patient and is most frequently diagnosed on the basis of a history of attacks of respiratory distress characterized by wheezing and the presence of classical râles. Three of the 7 patients observed gave histories which were suggestive of asthma and, in each of the 3, unilateral basilar wheezing was noted on auscultation. It is to be noted that in none of these patients was an excitant proved by allergic studies. Four patients of the group gave histories which were entirely negative for any factor which would suggest the possibility of asthma. One representative patient from each group has been chosen for presentation.

## CASE REPORTS

CASE 1.—A 43-year-old white man reported to the outpatient department with a history of episodes of coughing and wheezing for several years. Allergy studies on two occasions had failed to reveal an excitant. Auscultation of the chest revealed localized transient wheezes over the left base posteriorly. Routine posteroanterior and lateral chest films did not reveal any pulmonary abnormalities. Bronchoscopy revealed moderately inflamed granular mucosa in the lower left main-stem bronchus. No spasm or excessive secretion was noted. Bronchograms (Fig. 1, *A*, *C*, *E*) performed at this time were reported as demonstrating cylindric bronchiectasis involving the right middle and lower lobes and the left lower lobe. Medico-surgical chest conference disagreed with the diagnosis of bronchiectasis and requested a second bronchoscopic study. Bronchograms were repeated after twenty-one days. The second series of films (Fig. 1, *B*, *D*, *F*) were reported as follows: ''Cylindrical bronchiectasis previously described in both lower lung fields is no longer apparent on this examination.'' Immediately following the second bronchogram, this patient developed a generalized urticarial rash relieved by Pyribenzamine. This patient was placed on outpatient medical treatment for chronic asthmatic bronchitis.

CASE 2.—A 23-year-old white man was transferred to Brooke Army Hospital with a diagnosis of ''Severe chronic bronchitis, probably on basis of bronchiectasis.'' History revealed repeated colds and influenza as a child. In 1949, while serving in Japan, he developed an acute bronchitis requiring hospitalization. Slight productive cough persisted as a residual of this illness. Sputum had changed to a yellowish color four months prior to this hospital admission; the volume increased to one-half cup per day. His attacks of bronchitis were characterized by fever, chills, chest pain, and blood streaking of sputum. Response was prompt to antibiotic therapy. Allergic studies did not reveal any excitant. Routine chest films were negative for pulmonary pathology. Physical examination of the chest revealed fine moist râles over the right upper lung field and throughout the left lung field, posteriorly. Scattered expiratory wheezes were noted.

Bronchoscopy was essentially normal. Bronchograms (Fig. 2, *A*, *C*, *E*) revealed bronchial dilatation and were reported as consistent with bronchiectasis. Films of the repeat bronchography (Fig. 2 *D*, *B*, *F*), performed fourteen days later, reveal normal filling. This patient was discharged on a medical regimen for chronic bronchitis.

### BRONCHOGRAPHY TECHNIQUE

Oral intake was withheld for a period of 4 to 6 hours. Nembutal, 1½ grains, was administered orally 1½ hours prior to the bronchography.

Immediately before the procedure, the patient received 100 mg. Demerol and 1/150 grain atropine, intravenously. Topical anesthetization was accomplished by spraying a freshly prepared solution of 0 5 per cent Pontocaine solution with 4 drops of 1:1000 epinephrine per ounce Particular care was taken to anesthetize thoroughly the pyriform sinuses, the glottis, epiglottis, and vocal cords prior to instillation of the anesthetic agent into the trachea Anesthetization should not be hurried Two cubic centimeters were injected through the laryngotracheal opening into each side of the pulmonary tree and 2 c c. were deposited on the carina. Routine bronchoscopy was performed. Bronchoscopy was routinely carried out on new patients referred for study to obtain an accurate picture of the bronchial mucosa.

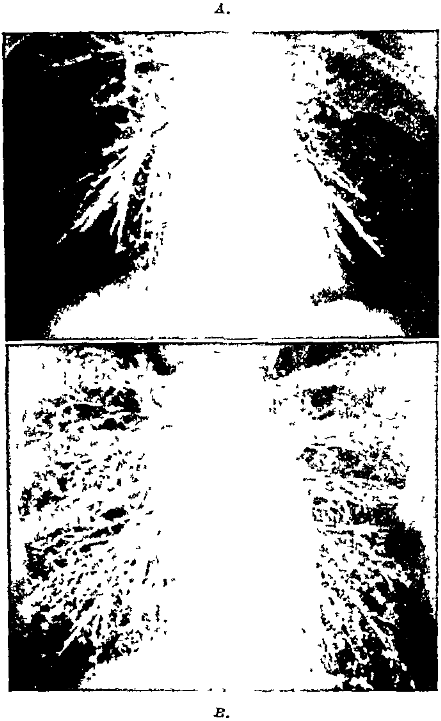


Fig 2—*A*, Initial posteroanterior bronchogram, revealing bilateral spasm *B*, Repeat posteroanterior bronchogram, normal pattern

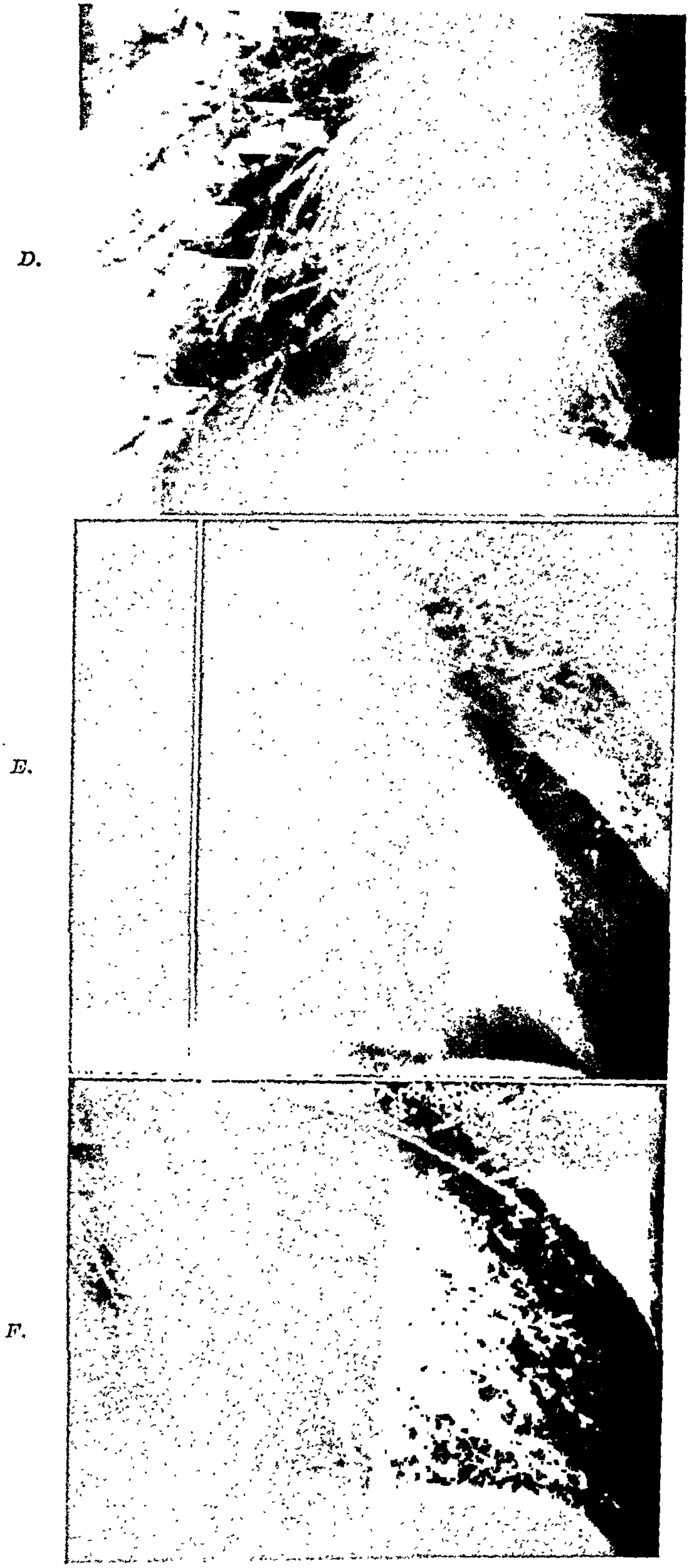


Fig. 1 (Cont'd).—*D*, Repeat left anterior oblique bronchogram, normal pattern. *E*, Initial right anterior oblique bronchogram, revealing spasm of lingula and left lower lobe. *F*, Repeat right anterior oblique bronchogram, normal pattern.

Approximately 5 c c of warm Lipiodol was injected during each of the four routine positions used for lobar filling The catheter was removed after the wet films were reviewed for adequacy of filling

Repeat bronchography, in the cases presented, was carried out by a technique altered to minimize (1) allergic response to drugs, (2) inadequate sedation, (3) inadequate anesthesia, and (4) trauma resulting from bronchoscopy

Sedation was increased to 3 grains Nembutal Demerol and atropine dosage was not increased Topical anesthesia was accomplished by spray technique using a Pontocaine HCl aerosol solution composed of 0 5 per cent Pontocaine, 2 0 per cent glycerine, 0 01 per cent Triton $H_2O$, and 0 01 per cent epinephrine This is essentially a detergent type of topical anesthetic which reduces the surface tension of secretions permitting a better contact between mucosa and anesthetic solution *A No 16 catheter was passed by indirect vision* technique Lipiodol in 22 c c volume was used as contrast medium

The second bronchography, utilizing increased sedation and improved topical anesthesia, gave normal bronchial patterns

### DISCUSSION

Typical cases of bronchospasm occurring during bronchography have been presented Prior to repeat bronchography, the probable causes of bronchospasm were considered and the technique altered accordingly Allergic response to drugs, inadequate sedation and anesthesia, and irritation of bronchoscopy were felt to be important factors in production of spasm From the standpoint of contact allergic response, Lipiodol cannot be considered the excitant as it was used in the second study, further, the usual reaction to iodine is usually a more generalized one Inadequate sedation is usually manifest by lack of cooperation and frequent coughing, which was not noted in any of these patients The more efficient topical anesthesia used in the second study was possibly of significance in eliminating a spastic element In no instance was initial bronchoscopy traumatic and certainly the areas of marked spasm were beyond the reach of the bronchoscope, in the secondary and tertiary divisions of the lobar bronchi

The phenomena of spasm during bronchography should be more generally appreciated by those who will be responsible for the diagnosis and treatment of patients with chronic cough Though such a picture might be anticipated in a small percentage of patients with an asthmatic history, it is of importance to recognize the bronchographic pattern of spasm in the nonasthmatic individual It seems advisable to review the basic points characteristic of both bronchospasm and bronchiectasis in order to alert the unwary to the possibility of confusing the two conditions

The cases presented clearly demonstrate the fallacy of a single bronchographic study and show how bronchospasm misinterpreted as bronchiectasis might easily lead to unwarranted excisional surgery

A No. 16 rubber catheter was passed through the lumen of the bronchoscope. As the bronchoscope was withdrawn, the distal end of the catheter was positioned just proximal to the carina.

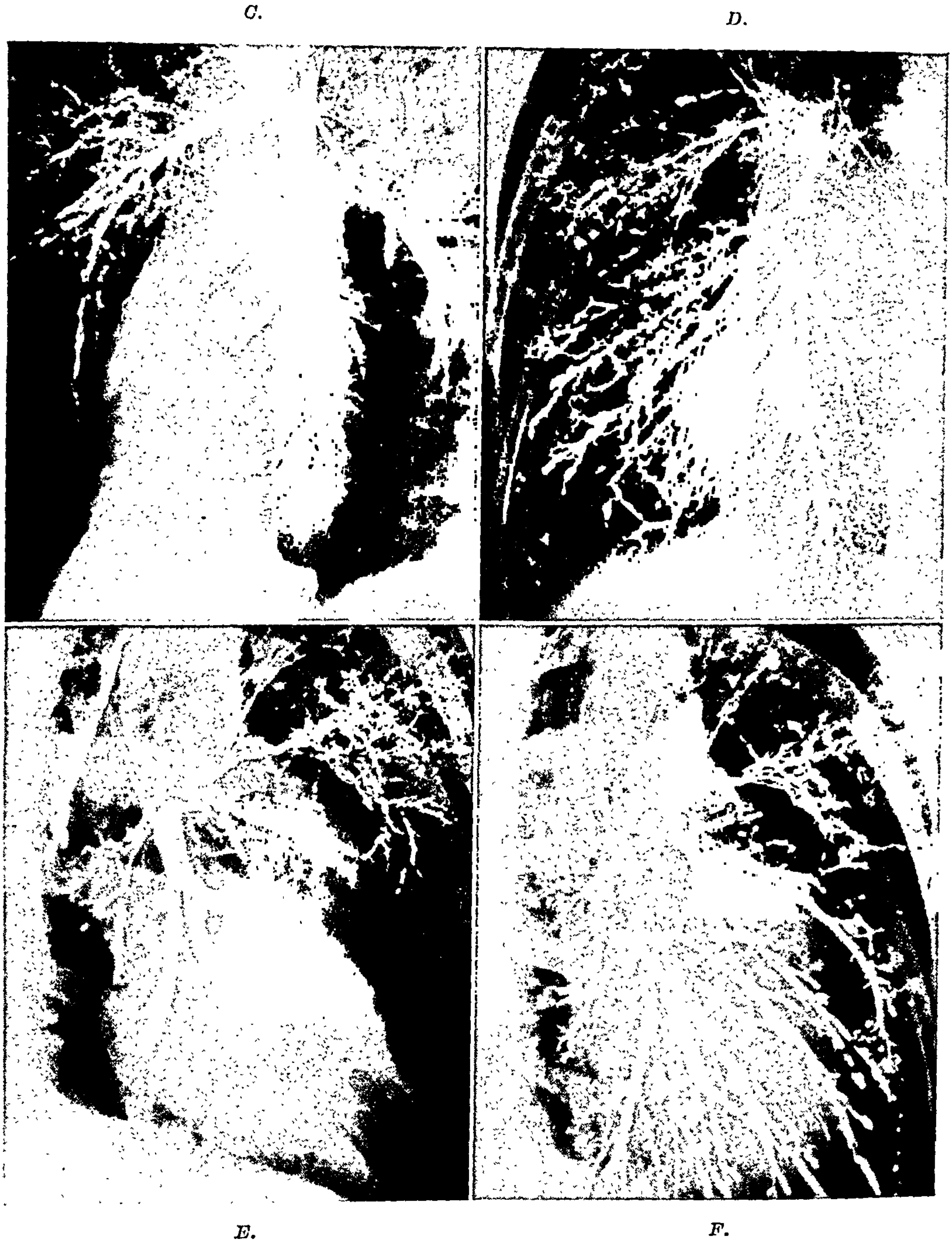


Fig. 2 (Cont'd).—*C*, Initial left anterior oblique bronchogram, revealing spasm right midle and lower lobes.

*D*, Repeat anterior oblique bronchogram, normal.

*E*, Initial right anterior oblique bronchogram revealing spasm left lower lobe and lingula.

*F*, Repeat right anterior oblique bronchogram, normal pattern.

# SIMULTANEOUS BILATERAL RESECTION WITH A SPACE DIMINISHING PROCEDURE FOR PULMONARY TUBERCULOSIS

VIKING OLOV BJORK, M D
STOCKHOLM SWEDEN

ALREADY in 1952, Overholt showed that simultaneous bilateral segmental resection could be performed In 1956, he reported ten such operations without mortality Lewis and associates had, by 1956, operated on 16 patients on both sides, simultaneously, without mortality So far, for pulmonary tuberculosis, only patients requiring no more than segmental resections on each side have been reported I have had 4 cases of pulmonary tuberculosis in which more extensive bilateral resections necessitated a space diminishing procedure as well All patients have tolerated well this rather large operation in one sitting The postoperative treatment must be carefully supervised

## TECHNIQUE

Lewis and his associates have recommended a supine position with the use of a transverse anterior incision through the third interspace, bilaterally and a transverse division of the sternum This incision seems convenient in their material where no more than segmental resections were required on each side The postoperative separation of the sternum in 3 cases of 16 will, however, make one hesitate to use this approach in tuberculosis Overholt's recommendation to use the face down position, with bilateral subcostal incisions, is preferable in most cases for the following reasons

1 After the resection on the first side, it may be found unwise to proceed with both resections simultaneously

2 After more extensive resection, it may be found desirable to perform a space diminishing procedure on one side It seems to be easier to perform an osteoplastic roof thoracoplasty or a diaphragm plastic in the face down position

It is important to avoid a narrow angle between the two incisions otherwise a necrosis of the skin will follow in that angle (Figs 1 and 2)

## CASE REPORTS

CASE 1—The patient, a 25 year old woman, had had bilateral cavitary tuberculosis for four years (Fig 3) The maximal breathing capacity was 62 L per minute Residual capacity was 31 per cent of the total capacity Bronchospirometry showed an oxygen uptake of 80 4 per cent and a ventilation of 73 7 per cent of the total on the right side

---

From Sabbatsberg Hospital Surgeon in Chief C Crafoord M.D Professor St Göran Hospital Physician in Chief A Westergren M D Professor Uttran Hospital, Physician in Chief N Levin M D

Received for publication April 13 1956

The bronchographic picture attributable to bronchospasm is characterized by the following features:

1. Bronchial dilatation of a hydrostatic nature occurs within the primary and portions of the secondary division bronchi.
2. Spasm of the tertiary and distal bronchi produces a horizontal obstruction to the flow of the contrast medium, consequently the spastic bronchi do not fill.
3. The normal space relations are maintained between the first and second division bronchi indicating the existence of aerated lung parenchyma.
4. Bronchiolar and alveolar filling is only patchy when evident distal to the zone of dilatation.
5. Proximal narrowing of a major bronchus is never visualized.

The following characteristics are essential to the bronchographic recognition of bronchiectasis:

1. Cylindric dilatation of a marked degree with complete loss of normal tapering tendency,
2. Saccular areas in direct continuity with the dilated bronchus,
3. Crowding of the bronchi resulting from decreased space occupied by pulmonary parenchyma of that involved area,
4. A decrease or complete absence of bronchiolar and alveolar filling, and
5. Proximal bronchial narrowing may or may not be evident.

The horizontal obstruction to flow evident in the initial bronchogram is not encountered in the markedly dilated bronchus of bronchiectasis. The maintenance of normal space relations between the several bronchi as determined by their position and direction in bronchospasm is not evident in bronchiectasis wherein the functionless parenchyma permits a crowding and downward depression of the bronchi. These particular points are basic in differentiation of bronchospasm and bronchiectasis.

## CONCLUSIONS

1. Repeat bronchography is essential in establishing the differentiating bronchial distention due to spasm from bronchiectasis.
2. Conference consideration of bronchograms is of value in preventing unnecessary surgery which might result from misinterpretation of a single bronchographic study.

to fibrotic changes in the lower lobe It was necessary to go between clamps around the cavitary disease in the left lower lobe In the remaining left basal segments, there were several small nodules Due to the relatively small amount of remaining lung tissue which was fibrotic and contained tuberculous nodules, I considered the addition of a left osteoplastic thoracoplasty in the same sitting a necessity The second to fifth ribs were resected posteriorly and then fixed to the sixth rib with nylon sutures through drill holes Immediately after completion of the operation, a tracheostomy was performed and the patient connected to an Engstrom respirator One day after the operation, she could take over ventilation herself for ten minutes and after nine days she did not need the help of the respirator any more

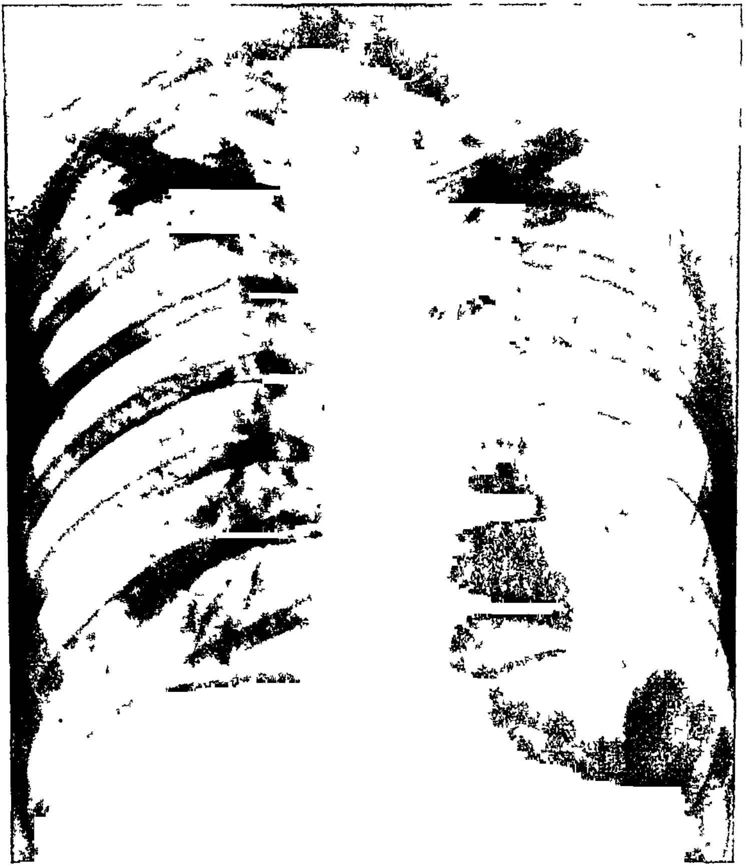


Fig 3 —Case 1 X ray of the lungs demonstrating large tuberculous cavities
lobes as well as in the superior segment of left lobe

The operation was performed in the face-down position. On the right side the upper lobe could be freed intrapleurally and then resected. The remaining lung parenchyma was of a good quality with only a few small hard nodules in the middle and lower lobes. When the right chest was closed, the left was entered through a subcostal incision under the fifth rib. It was necessary to remove the whole left upper lobe and the superior segment of the lower lobe in the extrapleural plane in one piece. Traction dissection could not be used due

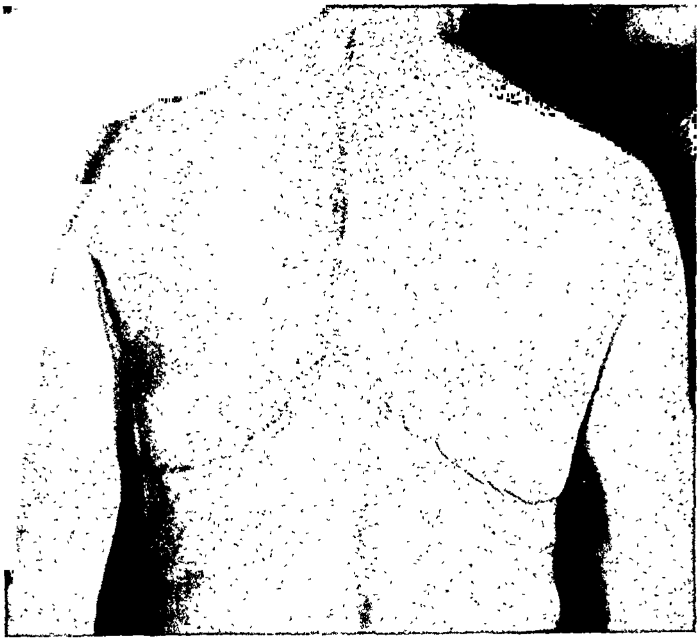


Fig. 1.—Photograph of the incision in Case 2. There was too sharp an angle between the two incisions resulting in a necrosis of the skin with a retarded healing.

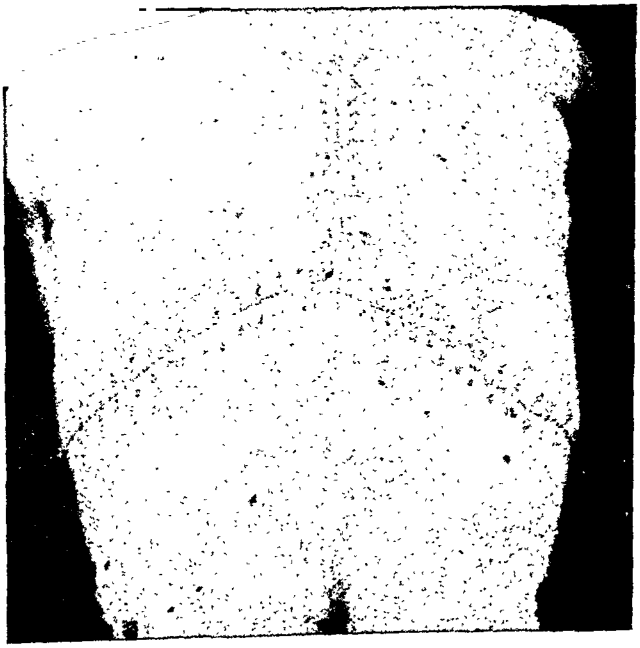


Fig. 2.—Photograph of the incision in Case 4. Here the incision was ideally placed and healed by primary intention.

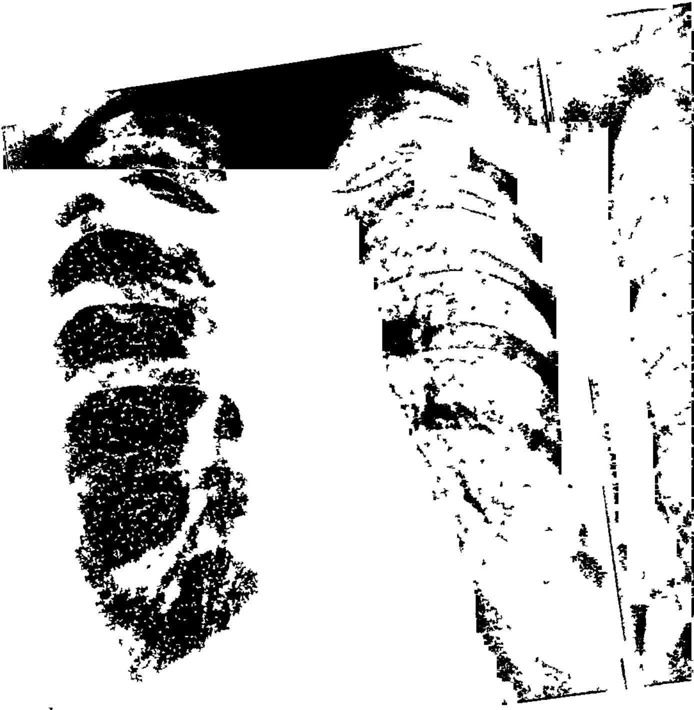


Fig 5

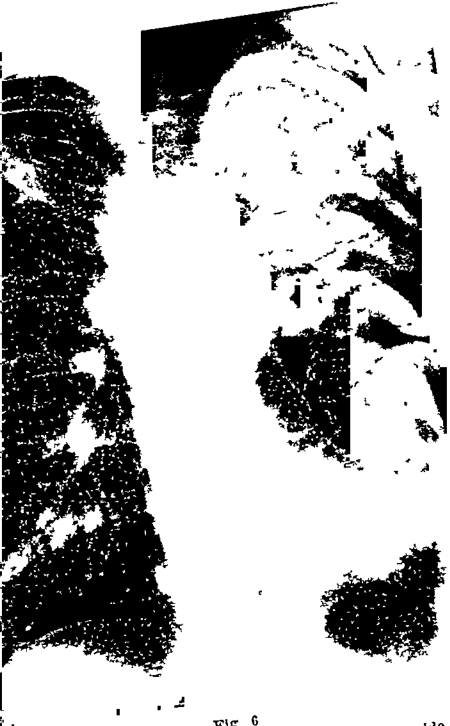


Fig 6

Fig 5 —Case 3 X-ray of the lungs demonstrating tuberculous cavities in both upper lobes and the lower lobe on left side

Fig 6 —Case 3 X-ray of the chest five months after simultaneous resection of the right upper lobe the apicoposterior and anterior segments of the left upper lobe as well as the superior and posterior basal segments of the left lower lobe and a diaphragm plasty on the left side

She regained strength rather slowly after the operation and had herpes zoster complicating the convalescence. One year after the operation she was in a very good general condition (Fig. 4).

CASE 2.—The patient, a 33-year-old man, had had bilateral cavitary tuberculosis for one year. His general condition was good. Maximal breathing capacity was 158 L. per minute. Bronchospirometry showed 53 per cent of the total ventilation on the right side.

At operation, the apicoposterior segment of the left upper lobe was removed. There were widespread nodules to be palpated in the lingula and in the lower lobe. An osteoplastic thoracoplasty with fixation of ribs 2 to 5 down to the sixth rib was added. The operation

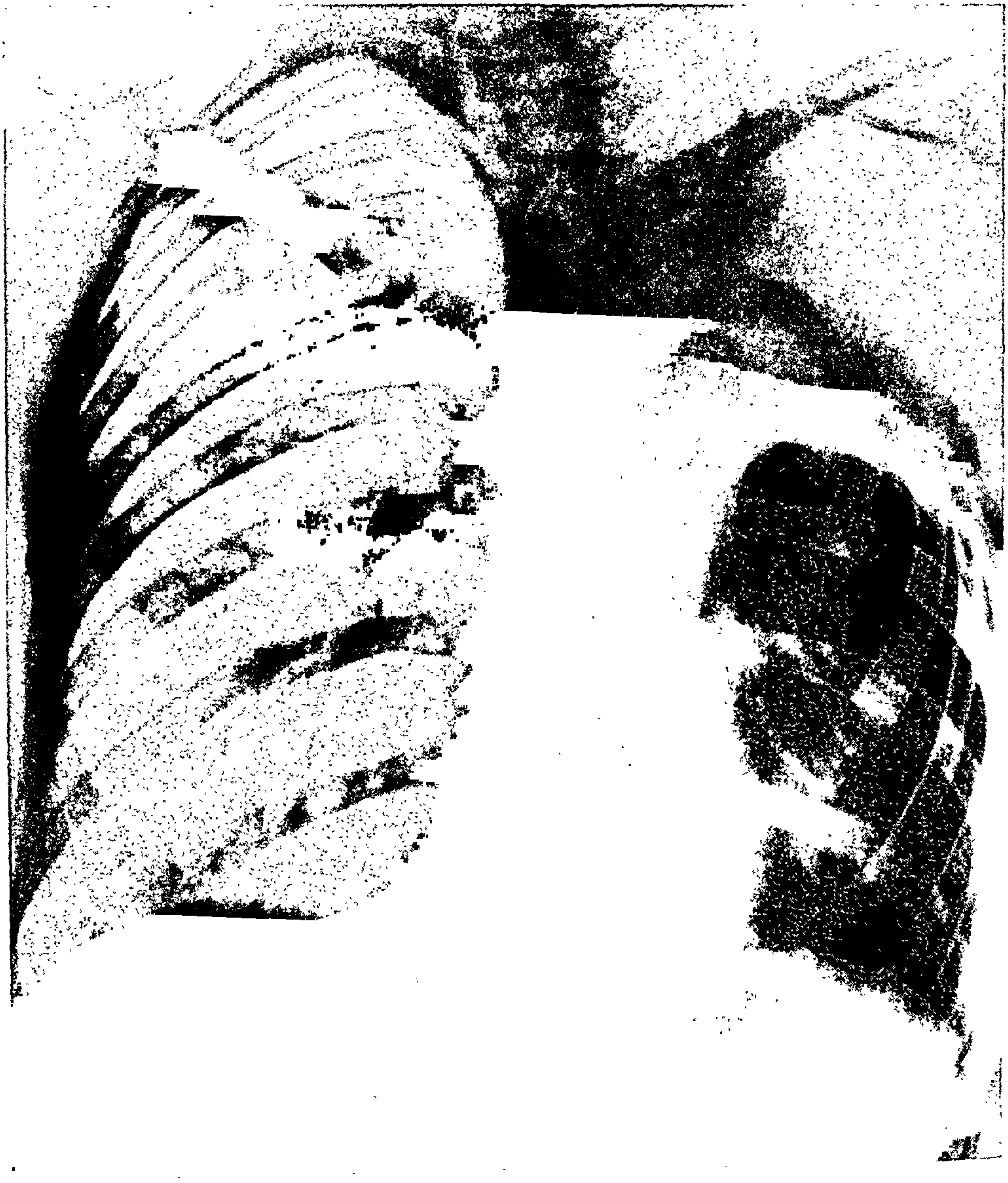
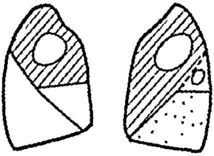
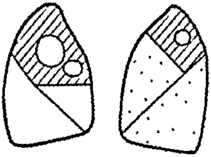
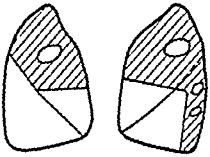
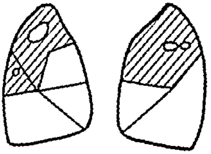


Fig. 4.—Case 1. X-ray of the lungs one year after simultaneous resection of the right upper lobe, left upper lobe, and superior segment of the left lower lobe, as well as an osteoplastic thoracoplasty on the left side.

TABLE I. SIMULTANEOUS BILATERAL RESECTION WITH SPACE-DIMINISHING PROCEDURES

| | | |
|---|---|---|
| Resection:<br>Right upper lobe + left upper lobe + superior segment left lower lobe + left osteoplastic thoracoplasty. Postoperative respirator treatment for 9 days. |  | Case 1. |
| Resection:<br>Apicoposterior segments left upper lobe + right upper lobe + left osteoplastic thoracoplasty. Spontaneous ventilation postoperatively. |  | Case 2. |
| Resection:<br>Right upper lobe + apicoposterior and anterior segments left upper lobe + superior and posterior basal segments left lower lobe + left diaphragm plasty. Postoperative respirator treatment for 24 days. |  | Case 3. |
| Resection:<br>Left apicoposterior and anterior segments + right apical and posterior segment upper lobe + superior segment right lower lobe + right diaphragm plasty. Spontaneous ventilation postoperatively. |  | Case 4. |

making the decision. The patient will gain several months in shortened hospital stay and have much less discomfort than in a staged procedure. Furthermore, the postoperative treatment of breathing exercises may be more difficult when cavitary tuberculosis remains on the contralateral side after operation.

Only carefully selected patients should be chosen for simultaneous bilateral resections. One need not hesitate to add a space-diminishing procedure at the same time. Naturally, this extensive pulmonary surgery should only be undertaken when the facility for postoperative respirator treatment is at hand.[5] It may be found necessary to relieve the patient from the respiratory work and ensure adequate ventilation for some time in the immediate postoperative period.

### SUMMARY

Four cases of simultaneous bilateral resection are reported. The resection has been extensive enough to warrant a space-diminishing procedure at the

was continued with a resection of the right upper lobe. The remaining parenchyma on the right side was in a good condition with only a few palpable nodules. The patient withstood the operation well and took over spontaneous ventilation immediately after the procedure.

CASE 3.—The patient, a 46-year-old woman, had had bilateral tuberculosis for twenty-five years. There had been a progression in the past year with bilateral cavitation (Fig. 5). The maximal breathing capacity was 79 L. per minute. The residual capacity was 30.5 per cent of the total capacity. Bronchospirometry showed 57.4 per cent of the total oxygen uptake on the right side.

At operation, the left side was first explored through a subcostal incision under the fifth rib. The apicoposterior and anterior segment of the left upper lobe as well as the superior and posterior basal segments of the left lower lobe were resected. Due to the rather extensive resection on the left side, it was decided to diminish that thoracic cavity. The diaphragm was therefore mobilized and resutured to the fifth intercostal muscle. Then the right chest was opened through a subcostal incision under the sixth rib. The whole right upper lobe was resected. Small nodules could be palpated in the lower lobe.

During the operation on the left side, an accident occurred. Some air entered the right side of the heart through the intravenous drip. The whole of the left pulmonary artery was full of air and its pressure rose considerably. The artery was immediately punctured by a wide bore needle and all air removed. After the evacuation of air, the pulmonary artery pressure decreased to 20 cm. water. After this experience, I think it is advisable to make a speedy opening of the chest and of the pulmonary artery in cases accidentally receiving too much air by an intravenous drip.

Two intrapleural tubes for drainage were left on each side. Immediately after the completion of the operation, a tracheostomy was performed and the patient connected to an Engström respirator. After two days, the patient could take over the ventilation for short intervals of ten minutes, but after twenty-four days she could definitely be without the respirator. The patient had a prolonged period of postoperative fever, but was able to leave the hospital two months after the operation in good condition (Fig. 6).

CASE 4.—The patient was a 43-year-old man who had had bilateral apical tuberculosis for sixteen years with bilateral cavitation for one year. The maximal breathing capacity was 98 L. per minute. Bronchospirometry showed 53 per cent of the total oxygen uptake and 48 per cent of the ventilation on the right side.

At operation, the right side was explored through a subcostal incision under the fifth rib. The apical and posterior segments of the right upper lobe and the superior segment of the right lower lobe were removed. The remaining lung parenchyma on the right side looked rather emphysematous. The diaphragm was mobilized and resutured to the fifth intercostal muscle bundle. Then the incision was extended to the left side and the apicoposterior and anterior segments of the left upper lobe were resected at the same operation. The patient made an uneventful recovery and had no difficulty in spontaneously maintaining an adequate ventilation.

## DISCUSSION

In cases of bilateral tuberculous cavitation where surgical treatment is being considered, a simultaneous resection should be seriously discussed. The addition of a space-diminishing procedure, either an osteoplastic thoracoplasty[2] or a diaphragm plasty[6] on one side, need not be a contraindication to a simultaneous bilateral resection. It is wise to explore the side with the more extensive disease first, using the face-down position. After completion of the first side it can then be decided whether it would be wise to proceed with the other side. Preoperative lung function studies with determination of the maximal breathing capacity and the oxygen uptake and ventilation on each side are helpful in

# PECTUS EXCAVATUM A SURVEY AND A SUGGESTION FOR MAINTENANCE OF CORRECTION

E. Harrison Griffin, M.D., and J. F. Minnis, Jr., M.D.
Brooklyn, N. Y.

BRAUHINUS,[1] in 1594, described the clinical condition of chonechondrosternon. Since then, this deformity has been recognized by various names: chonechondrosternon, trichterbrust, chondosternal depression, tunnel chest, and pectus excavatum, the first three being the more descriptively accurate, the last, the more commonly accepted term. Pectus excavatum may be described as a depression deformity of the body of the sternum and costal cartilages beginning at or inferior to the manubriogladiolar junction and reaching a maximum at the xyphoid. The classical picture is that of a "round shouldered, hollow chested, and pot bellied" child.[2]

The generally accepted incidence is 0.06 per cent, with males predominating by a ratio of four to one.[3]

A familial tendency has been demonstrated by Lester[5] and Sainsbury,[4] who believes that the defect is usually recessive, although, in one family, a dominant character was apparent. Most authors,[4-8] with the exception of Effler,[9] believe the deformity is congenital. Brown and Cook[10] reported associated anomalies in 31.8 per cent of their series of 44 cases. The consensus has been that pectus excavatum is a progressive disease,[6-13] however, Gross[14] has stated that "there are many babies in whom the deformity does not progress . . . ."

## ETIOLOGY

Various theories of historical interest regarding the etiology of pectus excavatum have been described by Ochsner and DeBakey:[3] arrested development of the sternum, hereditary syphilis, rickets, constitutional anomaly due to degenerative disease, poor posture, etc. Flesch[15] suggested, in 1873, that funnel chest was caused by an overgrowth of the ribs. As late as 1944, Sweet[16] and others gave some credence to this theory.

In 1939, Brown[13] presented a feasible explanation of the causal mechanism in his "substernal ligament" or "central tendon" theory, and opened a new approach to the surgical alleviation of this deformity. The substernal ligament has been designated as ligamentous fibers from the transversus thoracis and membrana sterni which merge posteriorly (retroxyphoid) to form the linea alba.[13] The central tendon of the diaphragm is a thin, strong aponeurosis located at the center of the diaphragmatic vault. It lies in close proximity to the xyphisternum and costal arch to which it is attached by rather short muscular

---

Received for publication April 16, 1956.

same operation. In 2 cases, an osteoplastic thoracoplasty and in 2 a diaphragm plasty were added. In 2 cases, the spontaneous ventilation was adequate, but in 2 it was necessary to add respirator treatment during the postoperative period. All 4 patients withstood the procedure well. A careful selection of patients is necessary.

## REFERENCES

1. Björk, V. O.: The Surgical Treatment of Pulmonary Tuberculosis, Acta Chir. scandinav. 107: 371, 1954.
2. Björk, V. O.: Thoracoplasty. A New Osteoplastic Technique, J. THORACIC SURG. 28: 194, 1954.
3. Björk, V. O., and Scrosoppi, F.: The Arterial Oxygen Tension After Resection With Simultaneous Osteoplastic Thoracoplasty, J. THORACIC SURG. 29: 105, 1955.
4. Björk, V. O.: Opportunité et modalités techniques de la réduction du volume thoracique après lobectomie pour tuberculose, Le Poumon, 8: 733, 1955.
5. Björk, V. O., and Engström, C. G.: The Treatment of Ventilatory Insufficiency After Pulmonary Resection With Tracheostomy and Prolonged Artificial Ventilation, J. THORACIC SURG. 30: 356, 1955.
6. Björk, V. O.: The Surgical Treatment of Lower Lobe Tuberculosis, J. THORACIC SURG. 31: 655, 1956.
7. Lewis, F. J., Shumway, N. E., Taufic, M., Zimmerman, B., Perry, J. F., Cohen, M., and Ring, D. M.: Simultaneous Bilateral Resection for Pulmonary Tuberculosis in Mental Patients, J. THORACIC SURG. 31: 93, 1956.
8. Overholt, R. H., Rheinlander, F. H., and Bolton, H. E.: Simultaneous Bilateral Pulmonary Segmental Resection, Bull. New England M. Center 14: 1, 1952.
9. Overholt, R. H.: Discussion of Lewis et al.[7]

## MANIFESTATIONS

The clinical manifestations of pectus excavatum are of three types: cosmetic, orthopedic, and cardiorespiratory. The patient presents with a flat, thin chest and a depression deformity of the thorax anteriorly, there is dorsal kyphosis, and the head is thrust forward. He, and especially she, is often shy and retiring due to the distorted habitus which provides a real psychologic problem.

The development or occurrence of cardiorespiratory symptoms are dependent upon four factors[10]: (1) position assumed by the heart, (2) total decrease in the anteroposterior diameter, (3) degree and shape of the depression of the sternal segments, and (4) age. Respiratory complaints may be noted at any age. During adolescence, the psychologic and orthopedic problems are prominent. So called "cardiac" symptoms become apparent during the second decade and are progressive with advancing years.

Lindskog[18] has stated that a minority of patients have symptoms resulting from the deformity. Most investigators report that a *majority* have respiratory complaints. This may be explained by impeded elevation of the ribs[10] and/or impaired action of the diaphragm[2, 17] which results in ineffectual cough and poor tracheobronchial toilet. Bronchiectasis may be a late complication of funnel chest.[21] Lester[17, 22] stated that the residual air was increased and the lung volume decreased (over 25 per cent) in proportion to the deformity but seldom to critical levels. Brodkin[6] likewise reported that vital capacity was diminished to as low as 50 per cent, however, in the series of Brown and Cook,[10] the vital capacity was essentially normal. They found the maximum breathing capacity to be the most valuable pulmonary function test: eleven of 40 patients showed a maximum breathing capacity of 50 per cent of the predicted normal and the average improvement following repair of the pectus excavatum was 31 per cent. Oxygen uptake is generally within normal limits as is oxygen saturation. Diminished maximum breathing capacity and/or electrocardiogram changes may be present even though cardiorespiratory symptoms are not apparent.[10] Since this disease is progressive, patients accommodate to the gradually impaired pulmonary function and therefore infrequently offer complaints referable to the dysfunction that exists. In some cases, there may be little or no actual limitation. Indeed, Manning[23] has reported the history of a Canadian prize fighter with definite pectus excavatum who presented no evidence of pulmonary or cardiac embarrassment.

Cardiac disease, other than that attributable to the thoracic deformity, has been reported as occurring "frequently."[10] Such an incidence would be dependent, in part, on the age groups included in the series. Ravitch,[11] Brodkin,[8] Gross[14] and others believe that cardiac symptoms are rare in infancy. Development of symptoms is dependent on: (1) pressure exerted directly on the heart, (2) cardiac compression, and (3) displacement of the heart, with possibly subsequent torsion or angulation of the great vessels. The pressure on the heart cannot be evaluated by the degree of sternal deformity. In a majority of instances the heart is displaced to the left and rotated posteriorly fibers. This point of attachment is shared with the substernal ligament. Therefore, any abnormal shortening of the anterior fibers and central tendon would cause depression of the lower end of the infant sternum, particularly during inspiration. Brown and Cook[10] have proposed that in funnel chest there is a neuromuscular imbalance whereby the anteroposterior fibers of the diaphragm are overstimulated. Asymmetrical deformity may be explained on the basis of overstimulation of one rather than both phrenic nerves.

Brodkin[8] has not been able to demonstrate the "retrosternal ligament" and proposes anomalous deformity of the diaphragm as etiological. The normal diaphragm is composed of "four major muscular divisions derived from the anterior septum transversum, the two lateral pleuroperitoneal membranes and the dorsal mesentery. Each, separated by a raphe, insert into the central trefoil tendinous portion. Contraction causes descent of the diaphragm without pull on any part of the chest wall. These contractions are responsible for the normal changes in development of the round thorax of the infant to the elliptical during the first year of life."[8] In chondrosternal depression deformity, the anterior portion of the diaphragm is tendinous showing a paucity of muscular fibers.[7] Retraction of the anterior chest wall during inspiration is produced by contraction of the diaphragm. This is the result of the pull of the tendinous attachment on the gladiolar-xyphoid junction of the sternum. Asymmetrical maldevelopment of the muscular diaphragm results in asymmetrical funnel chest.

Pectus excavatum, with paradoxical sternal retraction, may be noted at birth. Ravitch[5] has found it difficult to implicate the substernal ligament as entirely responsible because fetal respiration probably does not exert sufficient force to produce such a deformity. Other authors[2, 5, 9, 12, 17, 18] consider faulty function or development of the central tendon to be etiological.

A final etiological factor is that based on upper respiratory obstruction. First proposed by Holt,[19] and later supported by Hollinger,[20] it may be explained in the following quotation: "Shortening of the diaphragm is initiated as a result of forced respiratory efforts to overcome an obstruction somewhere between the carina and nose or mouth. The negative pressure thus produced causes a depression of the sternum because the cartilaginous portion of the chest is its weakest area. This leads to a contracture of the diaphragm which eventually becomes permanent."[19] It is generally recognized that respiratory obstruction may cause depression of the sternum with even marked paradox in infancy. We believe that this deformity is a separate entity and should not be confused with pectus excavatum. Probably this theory is only a contributory factor; however, Lester[2] has suggested that any obstruction to the respiratory tract be corrected prior to attempting reparative surgery for the pectus excavatum. Decrease in the anteroposterior diameter of the thorax, found in this deformity, impedes elevation of the ribs, and effectively prevents satisfactory clearing of the tracheobronchial tree.[10] Thus, an obstructive mechanism may be initiated secondary to costosternal defect.

costal cartilages were resected, turned over as a plate, and resutured to the rib ends Alexander[31] reported using a "T" shaped sternotomy in the treatment of a case of traumatic pectus excavatum in 1931 That same year, Sauerbruck[3] devised a plastic repair, resection of the fourth to eighth costal cartilages bilaterally and then elevating the body of the sternum by traction apparatus attached to an overhead frame In 1933, Carr[2] reported that Jerome Head[33] successfully performed an operation similar to that utilized by Sauerbruck, except that the body of the sternum was transected and then elevated by traction

With the publication of Brown's[13] classical description of the anatomic basis for and the surgical treatment of pectus excavatum, operative management of this deformity became established He proposed two operations, one for the incipient stage in infancy and the other for the fully developed stage The former has been termed the "simple" operation, advocated in children under the age of 2½ to 3 years The latter is known as the "radical" operation, utilized in older children and adults where the deformed costal cartilages have become fixed in the chondrosternal defect

The "simple" operation consists of simple division of the posterior ligamentous attachments to the sternum and xyphoid process As originally described[13] and presently widely utilized,[9 10 13 17 18 34] the xyphisternum and retrosternal ligaments are divided Following these procedures, if the cartilages show little or no deformity, the wound is closed without dividing cartilages or using traction apparatuses Variations have been described[8 10 17] but all follow the principle of separating the central tendon and retrosternal ligament from the body of the sternum Gross[14] has stated that he does not believe that simple division of the substernal ligament is sufficient to provide correction in more serious cases Lester[17] limits the use of this operation to infants under one year of age (only 2 have required further surgery), Brown and Cook[10] to those under 2 to 2½ years, and Brodkin[8] to those under 2½ years

The "radical" operation consists of the "simple" procedure plus three added steps (1) division, with or without resection, of the costal cartilages bilaterally, usually from the third to the eighth or ninth ribs, (2) transverse cuneiform osteotomy involving the anterior table of the sternum at a level with the third or second costal cartilage, and (3) elevation of the gladiolus Some form of this procedure has been almost universally adopted by American surgeons

The more prominent variations are listed

Brown and Cook[10] Resection of costal cartilages bilaterally, usually from the third to eighth, anterior transverse sternotomy, division of the xyphisternum and substernal ligaments, external wire traction elevation of the sternum in an overcorrected position

Brodkin[8] Resection of segments of cartilages from the costal arch to the second rib, the xyphoid process is excised, diaphragm is separated from the anterior attachment at the medial ends of the lowest cartilages and may be sutured to the free edge of the recti muscles, in complete anterior transverse

Hutcheson[21] has reported several cases in which the heart was markedly deviated into the left hemithorax without any evidence of embarrassment. Ravitch[11] has stated that arrhythmia resulted from direct pressure effects and that displacement or compression of the heart lead to mechanical interference with cardiac function. Circulation time and venous pressure are normal.[10] Brodkin[8] has been impressed with the relatively frequent presence of an orthostatic blood pressure and pulse, which have returned to normal postoperatively. The literature does not corroborate this finding. Often there is a systolic murmur which is probably functional.[22] The electrocardiogram demonstrates no specific type pattern. Most common findings are right axis deviation,[8, 11, 12, 18] sinus arrhythmia, low voltage, and inverted T waves in Leads III and IV.[8] Often these abnormalities will revert to normal postoperatively. Dorner[25] reported the case of a 28-year-old man who showed: supraventricular tachycardia with a 5:1 to 1:1 block, palpitation, right heart strain, peaked P waves, tricuspid murmur, and distortion of the tricuspid valve (angiocardiographically) presumably due to compression of the right atrium. Following plastic correction of the pectus excavatum, the electrocardiogram, chest x-ray, and angiocardiogram showed none of the previous abnormalities.

## MANAGEMENT

Nonsurgical methods have long been outmoded; belts and other such appliances have been discarded in the correction of pectus excavatum. Yet, as late as 1938, Cotton,[26] in his discussion of a paper by Truesdale,[27] strongly advocated breathing exercises rather than surgery.

The indications for surgery are cosmetic, orthopedic, and physiologic. Quite naturally, all patients have a cosmetic defect. If the disease is permitted to progress, most persons will become round-shouldered, the head will be thrust forward, and kyphoscoliosis may present a problem. It is apparent that every patient has *an* indication for surgery when a diagnosis of funnel chest is made. The only point of contention is *when* should he be subjected to surgical repair. Often the physiologic indications, primarily cardiorespiratory disturbances, dictate the time of intervention. Lester[17] has advocated "early" surgical repair if the patients "have or are about to develop symptomatic and functional disturbances." Brown and Cook[10] have proposed waiting until after the eighteenth month. Gross[14] has suggested that operation be deferred until the third or fourth year. In our review of the literature, the youngest patient reported was three months[11] and the oldest, 40 years of age.[2]

*Surgical Corrections.*—Ludwig Meyer,[28] operating in 1911, was probably the first to attempt a surgical correction of trichterbrust. His patient was a young adult having cardiac symptoms. The procedure consisted of resection of the second and third costal cartilages on one side. In 1913, Sauerbruck,[29] having a similar patient, successfully resected the right costal cartilages and anterior ribs from the fourth rib to the costal margin. The result was so satisfactory that he subsequently repeated this procedure on 3 patients. Hoffmeister,[30] in 1927, described the Lexter operation; the sternum and attached endotracheal anesthesia. Lester[17] has drawn particular attention to postoperative subglottic edema secondary to endotracheal intubation. Protection for the anterior chest wall, following removal of traction apparatus, probably is not a necessity; however, Gross[14] has suggested that a light plaster vest be worn for one month following discharge from the hospital.

The mortality rate is extremely low. Indeed, the recent literature is almost devoid of mention of operative deaths. Lester[1] reported one death in 94 operations. Ravitch[11] reported one death, from massive infection, in 21 operations. By contrast, Ochsner and DeBakey,[3] in their review of the literature in 1939 reported six deaths in 32 operative procedures.

## RESULTS

The over-all results of repair for funnel chest are good. In order to consider a result as satisfactory, there should be a correction of better than 75 per cent of the original defect.[17] Possibly the symmetrical deformity offers a better reparative prognosis than the asymmetrical.[14] On occasion, the cosmetic result may not be as satisfactory as the physiologic improvement. Brown and Cook[10] have stated that the "respiratory and psychologic manifestations" usually will be improved; however, the "cardiac" findings are often irreversible and show little change postoperatively even though there may be subjective improvement. The case, presented by Dorner and his associates[7] with severe cardiac findings, demonstrated an excellent response to operative correction.

The result which is apparent within the immediate postoperative period is a poor index as to the final outcome. During this time, the improvement is usually maximal, diminishes slowly, and finally stabilizes at about the fourth week.[10, 14] Therefore, final evaluation must be deferred until the second or third month, postrepair.

## CASE REPORTS

Six cases of pectus excavatum from the thoracic surgery services of the Methodist Hospital of Brooklyn and Kings County Hospital are presented.

TABLE I

| CASE NO. | AGE | SEX | CHIEF COMPLAINTS | OTHER ANOMALIES | ELECTROCARDIOGRAM | CARDIAC COMPRESSION |
|---|---|---|---|---|---|---|
| 1 | 6 | | *Deformity* | *Interventricular septal defect* | *Right axis deviation* | *No* |
| 2 | 8 | | Deformity | None | None | No |
| 3 | 11 | | Deformity and dyspnea | None | Left axis deviation, sinus arrhythmia | No |
| 4* | 14 | M | Deformity and chest pain on exertion | None | Sinus arrhythmia | Yes |
| 5† | 14 | M | Deformity and mild exertional dyspnea | None | Normal | No |
| 6 | 2 | M | Deformity | None | Sinus arrhythmia | No |

*Recurrence of pectus excavatum following former repair.
†Asymmetrical (right) deformity.

sternotomy; after elevation of the sternum, a costal cartilage free graft is placed beneath the lower end of the gladiolus, bridging the gap between the two hemithoraces anteriorly; all divided cartilages are sutured; no external traction apparatus is used because the sternum now rests on the cartilage graft.

Lindskog[12]: Resection of deformed costal cartilages; anterior transverse sternotomy; division of the diaphragmatic attachments to the xyphisternum without disarticulating the xyphoid process; elevation of the gladiolus in over-corrected position with wire suture traction.

Ravitch[11]: Resection of all re-formed costal cartilages; disarticulation of the xyphisternum, division of substernal ligament; anterior cuneiform osteotomy of the sternum as high as the most superior resected cartilage; the body of the sternum is elevated and held in this corrected position by silk sutures placed at the site of osteotomy; no traction apparatus is employed.

Gross[14]: Division of the xyphisternal articulation and substernal ligament; bilateral double wedge-excision of diseased costal cartilages; anterior transverse sternotomy and, occasionally, longitudinal sternotomy; elevation of the gladiolus and suture of wedged cartilages and sternum in the corrected position; external wire traction is used to maintain the sternum in its corrected position.

Dailey,[35] in 1948, was probably the first to utilize buried elevation support for the freed sternum rather than external traction. A rib graft was anchored to the anterior surfaces of the second right rib and the third left rib, passing beneath the elevated sternum. The result was quite satisfactory. This procedure was advocated for use in those patients who had attained major growth. Adams,[36] Woods, Overholt, and Bolton,[37] and Dorner, Keil, and Schissel,[25] later also utilized the substernal rib strut. Miscall[38] reported his unfortunate experience in the use of buried, crossed Kirschner wires for sternal elevation.

With his vast experience of 94 operations for pectus excavatum in 90 patients, Lester[17] has proposed three operations: "simple," "radical," and "modified" radical. The "simple" and "radical" procedures are essentially the same as described by Brown;[10, 13] the "simple" is reserved for infants under 12 to 14 months of age; the "radical" for those over 2½ to 3 years of age. Those infants who fall in the intervening age group are the ones for whom the "modified" operation was devised. It is similar to the "radical" procedure except that the third and fourth rib cartilages are not divided and the sternotomy is performed at a level with the fifth rather than the third rib.

## COMPLICATIONS

The most frequent operative complication is pneumothorax. Bleeding may be profuse; however, frank hemorrhage is quite uncommon.

Complications noted in the postoperative period are primarily those attendant with pneumothorax-atelectasis, pleural effusion, empyema. Paradoxical respiration may present a problem should there be inadequate stabilization of the sternum. Wound infection and keloid formation are sometimes found; the latter being encountered almost universally in longitudinal incisions over the sternum. There are also, those complications which are incident to

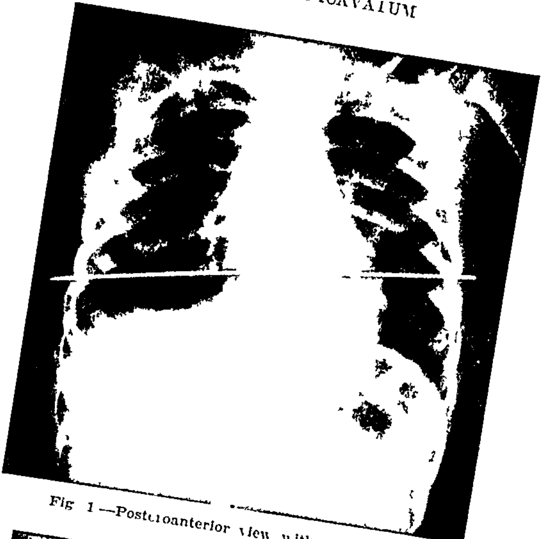


Fig 1 —Posteroanterior view with pin in situ

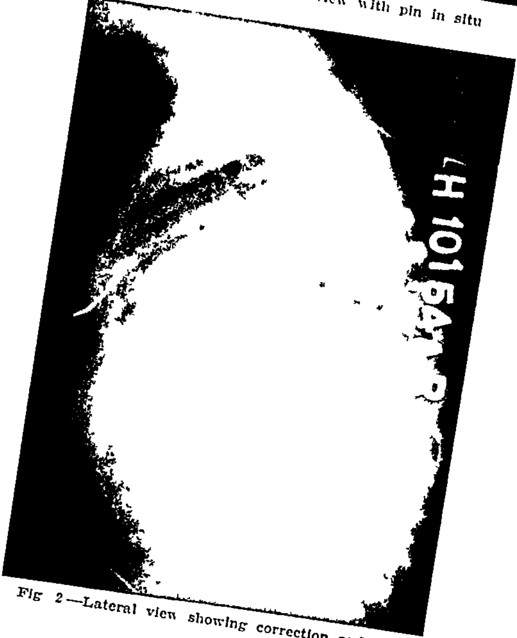


Fig 2 —Lateral view showing correction and pin

All patients were subjected to similar procedures utilizing the new method of sternum stabilization. It is not within the scope of this paper to offer case histories of a control group of patients by more conventional techniques.

The operative procedure followed in 5 cases was the classical "radical" one described by Brown[16] and Lester.[17] A midline incision was performed from the manubriogladiolar junction to just inferior to the tip of the xyphoid process. Muscular attachments to the costal cartilages were separated by sharp dissection. The xyphoid was divided from the gladiolus at the xyphisternal junction. Retrosternal muscular and ligamentous attachments were divided. Segments of all deformed costal cartilages were resected subperichondrially, from the costal arch to as high as the second rib. Transverse anterior cuneiform sternotomy was performed at a level with the highest resected cartilage. The sternum was elevated. A medium-sized Kirschner wire or long Steinmann pin was inserted between the anterior and posterior tables of the gladiolus at a level with the fourth or fifth costal cartilage. The pin was brought out, bilaterally, on the anterior surfaces of the fourth and fifth ribs, penetrating pectoral muscle and skin to emerge just below each nipple. Ordinary corks were threaded on the exposed pin, bilaterally, and the tips bent to prevent the corks from disengaging. Soft gauze was placed beneath the corks to afford protection of soft tissues against pressure effects. Collodion dressings covered the pin puncture sites. Strips of adhesion tape, from cork to cork, added further protection. Soft tissues were approximated in the midline. The anterior fascia of the rectus abdominis was sutured at the site of division from the lowest cartilages. Numerous sutures closed the subcutaneous tissues to provide less tension on the skin sutures in an attempt to avoid or minimize keloid formation. Drainage was utilized in a majority of cases; however, no wound complications were noted in any case. In 2 patients, the pleura was inadvertently entered during surgery with attendant pneumothorax as a complication which was readily controlled by intrapleural catheter closed drainage. The supporting pin was extracted, with minimal discomfort to the patient, on the second to third week postoperatively.

One patient (Case 5) with an asymmetrical deformity was subjected to a different operative procedure from the others. On the more deformed side, double wedge resections of cartilage were removed, modified after Gross.[14] On the other side, small segments were resected. Following transverse sternotomy and elevation of the gladiolus, wire sutures were placed at the sternotomy site and between reapproximated costal cartilages.

TABLE II

| CASE NO. | INDICATIONS FOR SURGERY | RIBS RESECTED | PIN EXTRACTED (DAY) | COMPLICATIONS | RESULTS |
|---|---|---|---|---|---|
| 1 | Cosmetic | 2 - 7 | 18 | None | Fair* |
| 2 | Cosmetic | 3 - 7 | 18 | None | Good |
| 3 | Cosmetic | 2 - 7 | 14 | Pneumothorax | Good |
| 4 | Cosmetic | 4 - 7 | 18 | Pneumothorax | Good |
| 5 | Cosmetic | 3 - 7 | 21 | None | Good |
| 6 | Cosmetic | 3 - 7 | 18 | None | Good |

*Resulted in overcorrection giving appearance of mild pigeon breast.

to the anterior chest wall[2, 7, 8, 10, 12-14, 17, 18, 22, 34, 30] accomplishes this requisite quite satisfactorily. There are, however, three inherent drawbacks; (1) the suture may break or tear out, (2) the patient must wear a rather cumbersome, though perhaps light, apparatus, and (3) the chest wall appliance may not remain stable. The rib strut method[17, 35, 36] is limited to use in adults. Miscall[38] utilized *crossed* Kirschner wires *retrosternally* for elevation correction of funnel chest in one case. One wire migrated into the pericardium several months postoperatively; however, the costosternal deformity had been successfully treated.

Kirschner wires or Steinmann pins, used as we have described, have the disadvantage of not maintaining overcorrection; if, indeed, this is a disadvantage. Our limited experience does not indicate that overcorrection is necessary. There is no danger of migration of the pin or of trauma to intrathoracic viscera because the pin remains separated from these structures. Danger of infection is no greater than with any other traction mechanism extending to the exterior by penetrating skin. The major advantages are; (1) the pin cannot disengage the sternum, as with wire sutures (assuming that the correct caliber pin is selected), (2) there is no cumbersome external appliance, only two corks, and (3) it is readily inserted without resort to additional incisions, as when rib graft struts are employed.

## SUMMARY

1. The etiology and evolution of the surgical treatment of pectus excavatum has been presented.
2. A new method of maintenance of the elevated sternum in corrected position has been described.
3. Six cases of pectus excavatum treated surgically are added to the literature.
4. We believe that no single operative technique is applicable to all patients with this deformity. Each case should be evaluated in the light of which particular procedure will lend itself most advantageously to the deformity at hand; in short, a custom-tailoring procedure.

## REFERENCES

1. Brauhinus: Cited in Ochsner and DeBakey.[3]
2. Lester, C. W.: Funnel Chest: Its Cause, Effects, and Treatment, J. Pediat. 37: 224, 1950.
3. Ochsner, A., and DeBakey, M.: Chone-chondrosternon. Report of a case and review of the literature, J. THORACIC SURG. 8: 469, 1939.
4. Sainsbury, H. S. K.: Congenital Funnel Chest, Lancet 2: 615, 1947.
5. Ravitch, M. M.: The Operative Treatment of Pectus Excavatum, Ann. Surg. 29: 429, 1949.
6. Brodkin, H. A.: Congenital Chondrosternal Depression (Funnel Chest) Relieved by Chondrosternoplasty, Am. J. Surg. 75: 716, 1948.
7. Brodkin, H. A.: Congenital Chondrosternal Prominence (Pigeon Breast). A New Interpretation, Pediatrics 5: 286, 1949.
8. Brodkin, H. A.: Congenital Chondrosternal Depression (Funnel Chest). Its Treatment by Phrenosternolysis and Chondrosternoplasty, Dis. Chest 19: 288, 1951.
9. Effler, D. B.: Pectus Excavatum: Surgical Treatment, Cleveland Clin. Quart. 20: 353, 1953.
10. Brown, A. L., and Cook, O.: Cardio-Respiratory Studies in Pre and Post-Operative Funnel Chest (Pectus Excavatum), Dis. Chest 20: 378, 1951.

## DISCUSSION

The patients presented varied from 2 to 14 years of age. All gave a history of upper respiratory infections or some form of respiratory distress. Only one patient had pulmonary function tests (Case 5); the significant abnormalities being a vital capacity of 65 per cent of predicted normal and a maximum breathing capacity of 77 per cent of the predicted normal. The electrocardiogram showed right axis deviation in one patient and sinus arrhythmia in 3. The indication for surgery in all patients was primarily "cosmetic." Five of the 6 cases were primary repairs; Case 4 was a patient who had been subjected to corrective surgery three to four years previously and had had a recurrence. Follow-up extends from three months to three years, postoperative. Results are considered "good" in 5 and "fair" in one. The reason for classifying one patient as having only a "fair" result is that the defect was overly corrected; producing, in essence, a mild form of pectus incarnatum.

Fig. 3.—The suggested method of maintenance of correction in an ambulatory patient.

## COMMENT

Maintenance of the elevated gladiolus in a corrected, or overly-corrected position is, in our opinion, inimicable to the success of reparative surgery for pectus excavatum. Wire traction sutures attached to an apparatus applied

# THE SURGICAL MANAGEMENT OF LARGE INTRATHORACIC GOITERS. CONTRIBUTIONS OF ANGIOCARDIOGRAPHY

LAURENCE MISCALL, M.D., RICHARD B. NOLAN, M.D., NATHANIEL FINBY, M.D.,
AND ISRAEL STEINBERG, M.D.
NEW YORK, N.Y.

LARGE tumors of thyroid origin are seen frequently in the superior mediastinum. Prompt surgical excision is indicated because of progressive symptoms due to displacement or obstruction of adjacent vital structures and the increasing difficulties inherent in delay. The reports of incomplete operation, postoperative hemorrhage, and infection with the use of the usual collar incision have been common enough to indicate that greater success may be achieved by the additional use of some type of thoracic incision. Experience has shown that this is not only safer in many cases but also indispensable in others. Furthermore, the very extensive use of tomography and angiocardiography at the New York Hospital demonstrates that these methods as supplements to other routine roentgenographic studies often yield information useful in the better adaptation of the operative approach to the anatomic situation of the tumor. These studies of 5 patients with very large thoracic thyroid tumors were selected to show how thorough roentgenographic study, especially angiocardiography, aided in planning a suitable surgical approach.

## CASE REPORTS*

CASE 1.—A 32 year old man was admitted to the hospital because an asymptomatic superior mediastinal mass had been found on a survey chest x ray. It was barely palpable in the supraclavicular space. The basal metabolic rate was -21 and other laboratory findings normal. On chest roentgenograms (Fig 1, *A* and *B*), a large right superior mediastinal mass extended inferiorly to the right upper lobe bronchus and displaced the trachea and esophagus to the left. Angiocardiography showed that the tumor also displaced the innominate artery and right brachiocephalic vessels to a right anterolateral position (Fig 1, *C* and *D*). A right anterior transthoracic approach combined with the usual cervical collar incision was selected.

*Operation.*—On Feb 16, 1949, the neck was explored through a low collar incision. Since the tumor could not be safely mobilized through this approach, the right thorax was opened anteriorly through the bed of the third rib. After incision of the mediastinal pleura, the tumor was freed without difficulty. With careful manipulation in both the chest and neck and with anterior retraction of the great vessels, the tumor was delivered through the thoracic inlet. A thin stalk connected it to the right thyroid lobe and after division and ligation of the superior thyroid vessels, the mass and right lobe were excised. The chest and neck were both drained and the wounds closed without repair of the mediastinal pleura.

*Pathologic Diagnosis.*—Nodular thyroid with involution and cystic degeneration.

---

From the Private Patient Surgical Service and the Department of Radiology, New York Hospital Cornell Medical Center and Triboro Hospital, New York, N.Y.

Received for publication April 24, 1956.

*General anesthesia administered by endotracheal technique was employed in all patients.

11. Ravitch, M. M.: Pectus Excavatum and Heart Failure, Surg. 30: 178, 1951.
12. Lindskog, G. F., and Felton, W. L., II.: Considerations in the Surgical Treatment of Pectus Excavatum, Ann. Surg. 142: 654, 1955.
13. Brown, A. L.: Pectus Excavatum (Funnel Chest)—Anatomic Basis; Surgical Treatment of the Incipient Stage in Infancy; and Correction of the Deformity in the Fully Developed Stage, J. THORACIC SURG. 9: 164, 1939.
14. Gross, R. E.: The Surgery of Infancy and Childhood, Philadelphia, 1953, W. B. Saunders Company, chapt. 57, pp. 753.
15. Flesch, M.: Ueber eine Seltene Missbildung des Thorax, Virchows Arch. f. path. Anat. 57: 289, 1873.
16. Sweet, R. H.: Pectus Excavatum, Ann. Surg. 119: 922, 1944.
17. Lester, C. W.: Pigeon Breast, Funnel Chest, and Other Congenital Deformities of the Chest, J. A. M. A. 156: 1063, 1954.
18. Lindskog, G. F., and Felton, W. L., II.: Pectus Excavatum. A Report of Eight Cases With Surgical Correction, Surg., Gynec. & Obst. 95: 615, 1952.
19. Holt, L. E. Jr.: Cited in Lester.[22]
20. Hollinger, P. H.: Cited in discussion of Lester.[39]
21. Lindskog, G. F., and Leibow, A. A.: Thoracic Surgery and Related Pathology, New York, 1954, Appleton-Century-Crofts, Inc., chapt. 2, pp. 28.
22. Lester, C. W.: The Surgical Treatment of Funnel Chest, Ann. Surg. 123: 1003, 1946.
23. Manning, G. W.: A Case of Funnel Chest, Canad. M. A. J. 53: 550, 1945.
24. Hutcheson, J. M.: Cardiac Complications of Funnel Chest, South. Med. & Surg. 101: 266, 1939.
25. Dorner, R. A., Keil, P. G., and Schissel, D. J.: Pectus Excavatum. Case Report With Pre- and Postoperative Angiocardiographic Studies, J. THORACIC SURG. 20: 444, 1950.
26. Cotton, F. J.: Cited in discussion of Truesdale.[27]
27. Truesdale, P. E.: A New Method of Dealing With Funnel Chest, New England J. Med. 218: 102, 1938.
28. Meyer, L.: Cited in Ochsner and DeBakey.[3]
29. Sauerbruck, E. F.: Die Chirurgie der Brustorgane, ed. 3, Berlin, 1928, G. Springer, pp. 735.
30. Hoffmeister, W.: Operation der Angeborne Trichterbrust, Beitr. z. klin. chir. 141: 214, 1927.
31. Alexander, J.: Traumatic Pectus Excavatum, Ann. Surg. 93: 489, 1931.
32. Carr, J. G.: The Cardiac Complications of Trichterbrust, Ann. Int. Med. 6: 885, 1933.
33. Head, J.: Cited by Carr.[32]
34. Lester, C. W.: Deformities of the Thorax of Congenital or Developmental Origin, J. Pediat. 42: 195, 1953.
35. Dailey, J. E.: Repair of Funnel Chest Using Substernal Osteoperiosteal Rib Graft Strut. Report of a Case With Four Year Follow-Up, J. A. M. A. 150: 1203, 1952.
36. Adams, H. D.: Costosternoplasty With Rib Strut Support for Funnel Chest in Adults. Surgical Practice of the Lahey Clinic. Philadelphia, 1951, W. B. Saunders Company.
37. Woods, F. M., Overholt, R. H., Bolton, H. E.: Pectus Excavatum, Dis. Chest. 22: 274, 1952.
38. Miscall, L.: Cited in discussion of Dorner, Keil, and Schissel.[25]
39. Lester, C. W.: Funnel Chest and Allied Deformities of the Thoracic Cage, J. THORACIC SURG. 19: 507, 1950.

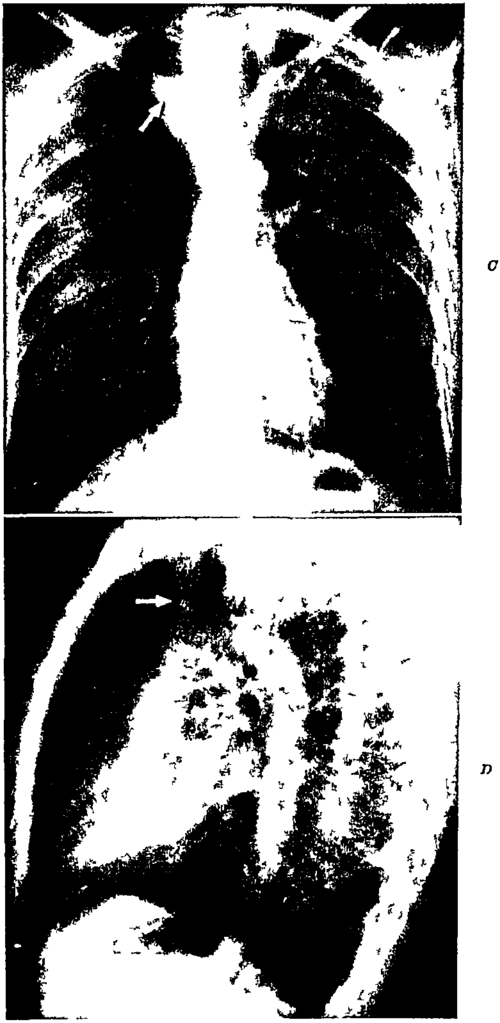


Fig 1 (Cont d) —*C*, Frontal angiocardiogram demonstrates the opacified left ventricle aorta and brachiocephalic branches Note that the tumor lies between the displaced trachea and innominate artery (arrow) *D* Lateral angiocardiogram also reveals the opacified left cardiac chambers and aorta. Note the forward displacement of the innominate artery (arrow)

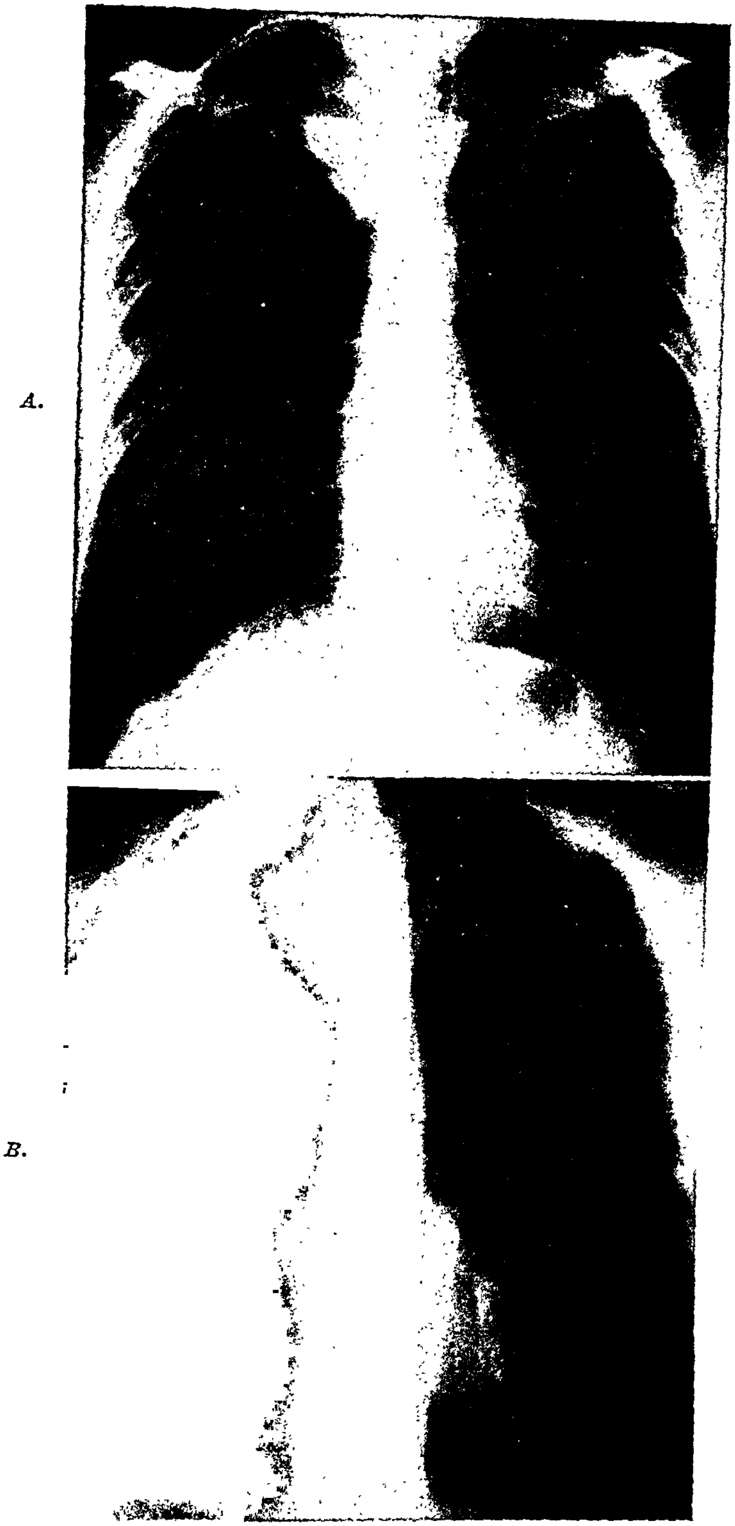


Fig. 1.—Case 1. *A*, Frontal chest x-ray shows a large right superior mediastinal tumor displacing the trachea to the right. *B*, Frontal tomogram demonstrates that the mass extends deep into the region of origin of the right bronchus, the trachea is not narrowed; the cartilaginous rings are intact.

CASE 2.—The patient, a 45 year old housewife, had had an enlarged thyroid gland for several years. Six months prior to admission she had noted progressively frequent and severe "smothering attacks." An enlarged nodular thyroid gland was present. The chest roentgenograms showed a huge right superior mediastinal mass extending posterior to the trachea and the esophagus, displacing them into the left hemithorax. Angiocardiography showed marked anterior and right lateral displacement of the right brachiocephalic vessels (Figs. 2 *A*, *B*, and *C*). In view of its size and intimate relation to the posterior mediastinal structures, a right thoracic approach combined with a collar incision was selected.

*Operative Findings.*—On Feb. 7, 1955, using a submammary incision, the right thorax was opened through the bed of the second rib. The tumor displaced the great vessels anteriorly and to the right, and extended from the right upper lobe bronchus to the thoracic inlet. It extended anteriorly and posteriorly around the trachea and esophagus. The pleura was incised and, following mobilization of the great vessels anteriorly, the tumor was separated from the bronchus and trachea. Traction on the tumor delivered the esophagus which was then freed under direct vision. Ligation of many small vessels was necessary. The size of this thoracic tumor would not permit passage into the neck and accordingly most of it was excised. Further dissection through a low collar incision permitted the remainder to be delivered into the neck and removed. The wounds were closed with drainage of the right chest only.

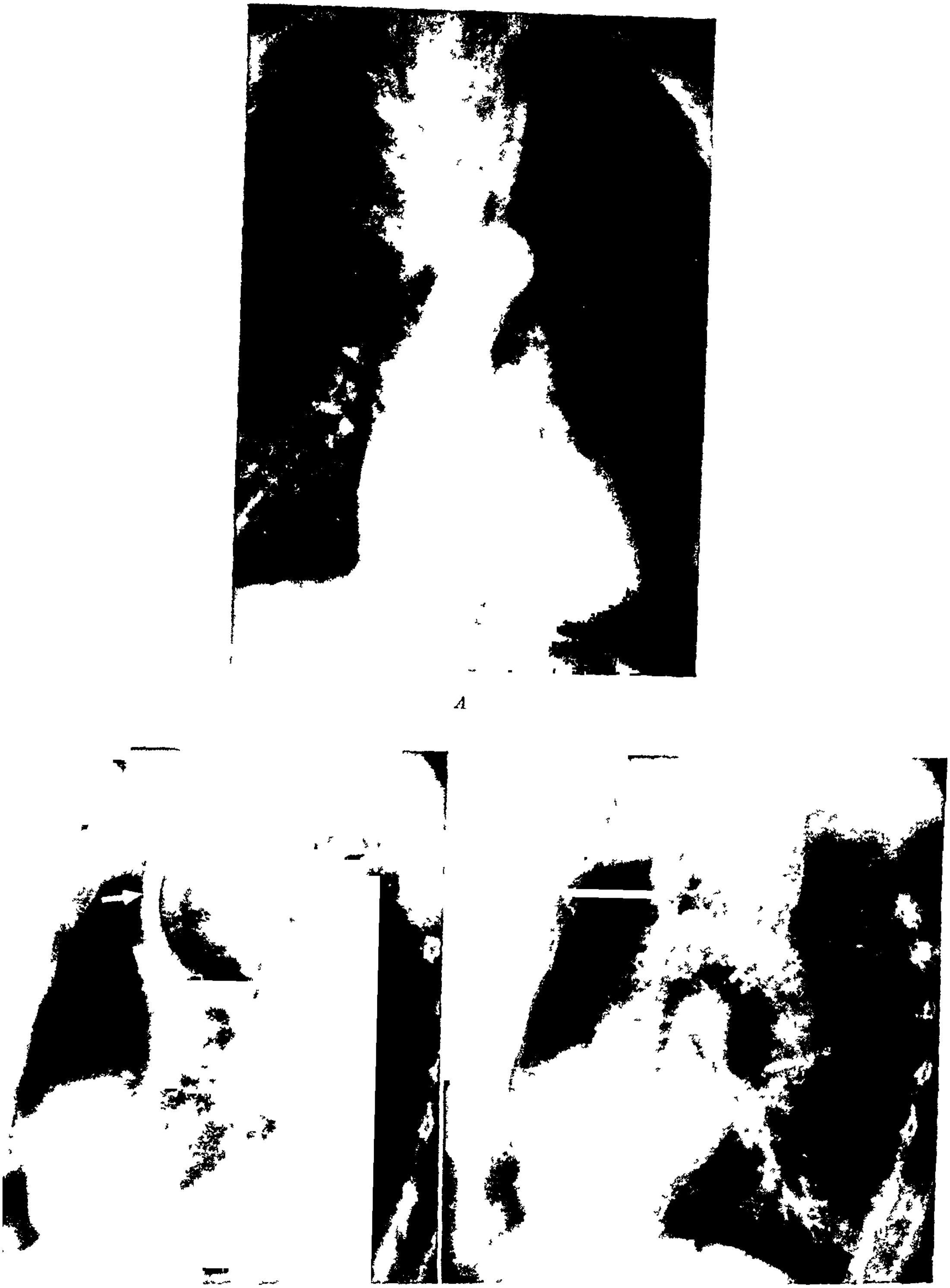
*Pathologic Diagnosis.*—Nodular cystic goiter with involution.

CASE 3.—A 64 year old housewife was admitted to the hospital with complaints of chest pain, dyspnea, and hoarseness of four months' duration. Through a cervical incision, a previous attempt at subtotal thyroidectomy had been unsuccessful. Roentgenography with tomography revealed a large right superior mediastinal mass which extended retrosternally and yet the esophagus and trachea were displaced toward the right (Fig. 3, *A*). Angiocardiography (Fig. 3, *B* and *C*) showed a compressed right innominate vein, right posterior displacement of the innominate artery, and displacement of the left brachiocephalic vessels to the left. The need for exposure of both sides of the trachea in the area between the great vessels was evident and therefore a sternal splitting incision in combination with a low collar incision was selected.

*Operation.*—On Feb. 28, 1955, the sternum was split from notch to xiphoid. The substernal portion of the tumor filled the mediastinum, extending inferiorly over the pericardium, displacing the superior vena cava anteriorly and to the right, and the innominate artery and branches posteriorly and to the right. The superior vena cava and innominate veins were mobilized, the left being compressed and barely visible on the anterior surface of the tumor. It was then possible to dissect the aorta and trachea free from the posterior surface. The connection of the tumor mass to the left thyroid lobe was divided and the mass excised. The trachea and esophagus were still markedly displaced to the right by a second mass lying posterior to the axis of the aortic arch and left brachiocephalic vessels. With retraction of these vessels to the left and inferiorly, this mass was mobilized to the thoracic inlet where it also was connected to the left thyroid lobe. Dissection in both the mediastinum and neck permitted the tumor to be delivered through a collar incision into the neck and excised with the left thyroid lobe. Both pleural spaces were drained.

*Pathologic Diagnosis.*—Nodular goiter with cystic degeneration and involution.

CASE 4.—The patient, a 64 year old housewife, complained of increasing cough, hoarseness, dyspnea, and headache for six months. The thyroid was nodular and symmetrically enlarged and engorged veins of the neck failed to empty normally. The chest roentgenograms (Fig. 4, *A* and *B*) revealed a massive superior mediastinal tumor, containing multiple amorphous calcific densities, which extended into the neck. The midline trachea was markedly and concentrically narrowed at the thoracic inlet productive of a "crowing" respiration



A

B.

C.

Fig 2.—Case 2 *A*, Frontal angiocardiogram shows the opacified left heart and aorta; huge goiter lies behind the trachea and is displacing the distal part of the innominate tery. *B*, Lateral angiocardiogram shows forward displacement of the right innominate vein arrow); the tumor extends deep within the mediastinum and is behind the trachea *C*, Lateral angiocardiogram shows the opacified left heart chambers and aorta Note the displacement of the brachiocephalic vessels

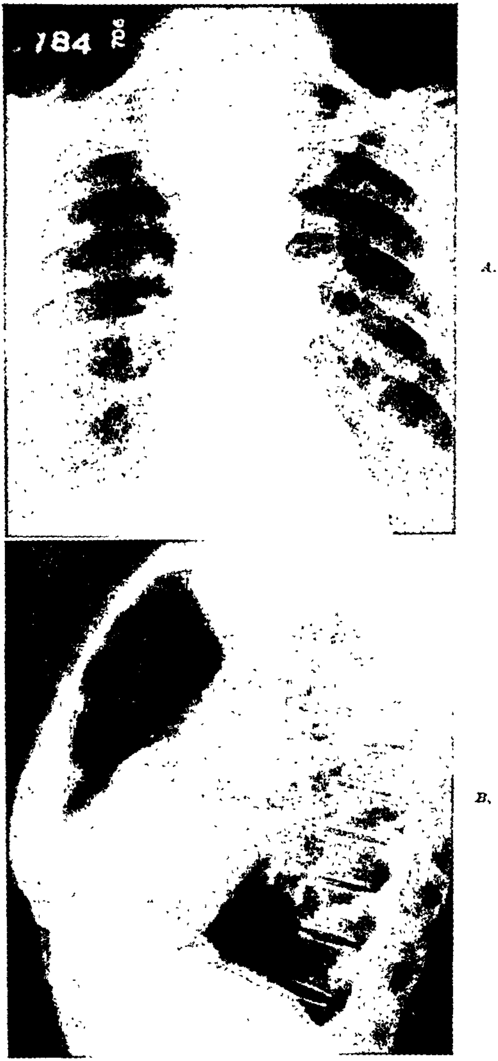


Fig. 4.—Case 4. *A*, Frontal chest x-ray reveals a massive superior mediastinal mass. *B*, Lateral x-ray shows that the mass surrounds and compresses the trachea and contains calcific deposits.

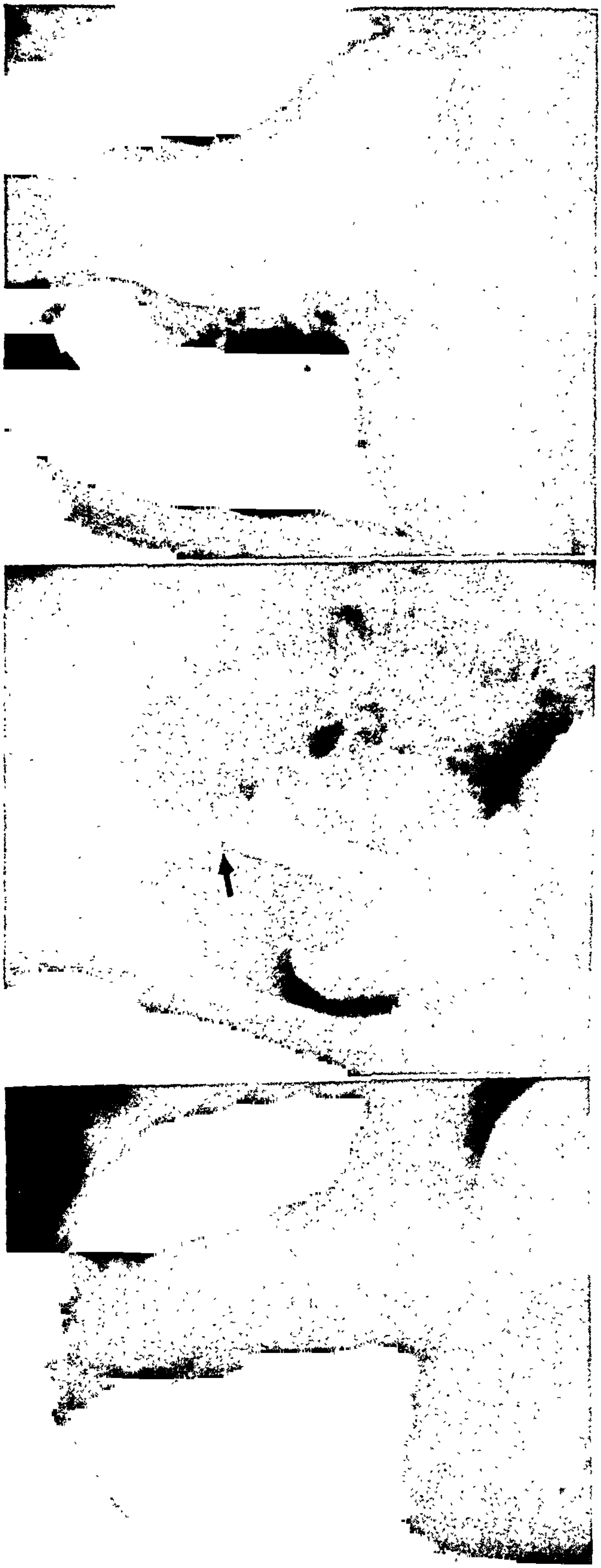
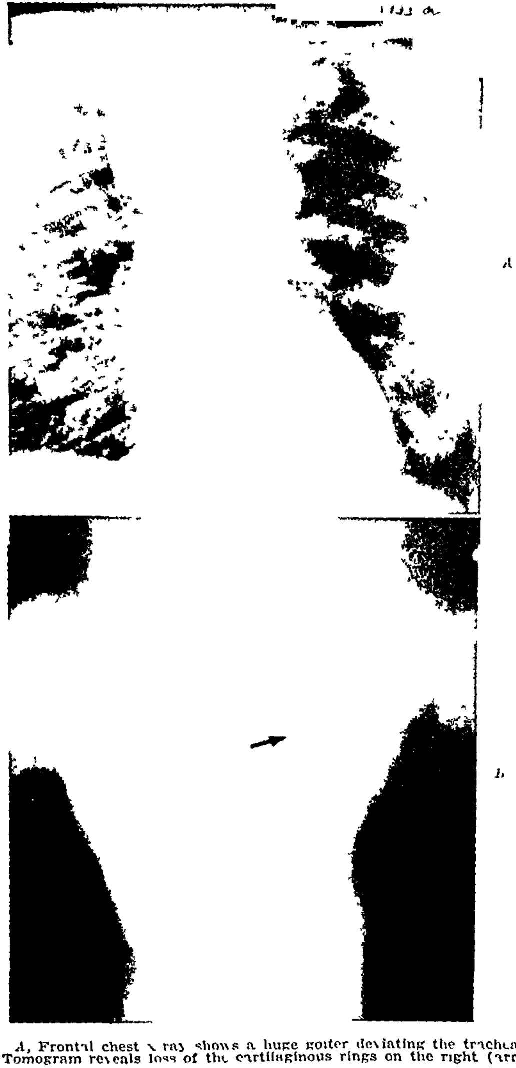


Fig. 3.—Case 3. *A*, Frontal tomogram demonstrates marked bowing of the trachea to the right by a huge goiter. *B*, Lateral angiocardiogram shows compression of the right innominate vein (arrow). *C*, Frontal angiocardiogram reveals opacification of the left heart and aorta. The right innominate artery (arrow) is markedly displaced by the goiter.



ig 5 —Case 5 A, Frontal chest x ray shows a huge goiter deviating the trachea toward the left B, Tomogram reveals loss of the cartilaginous rings on the right (arrow).

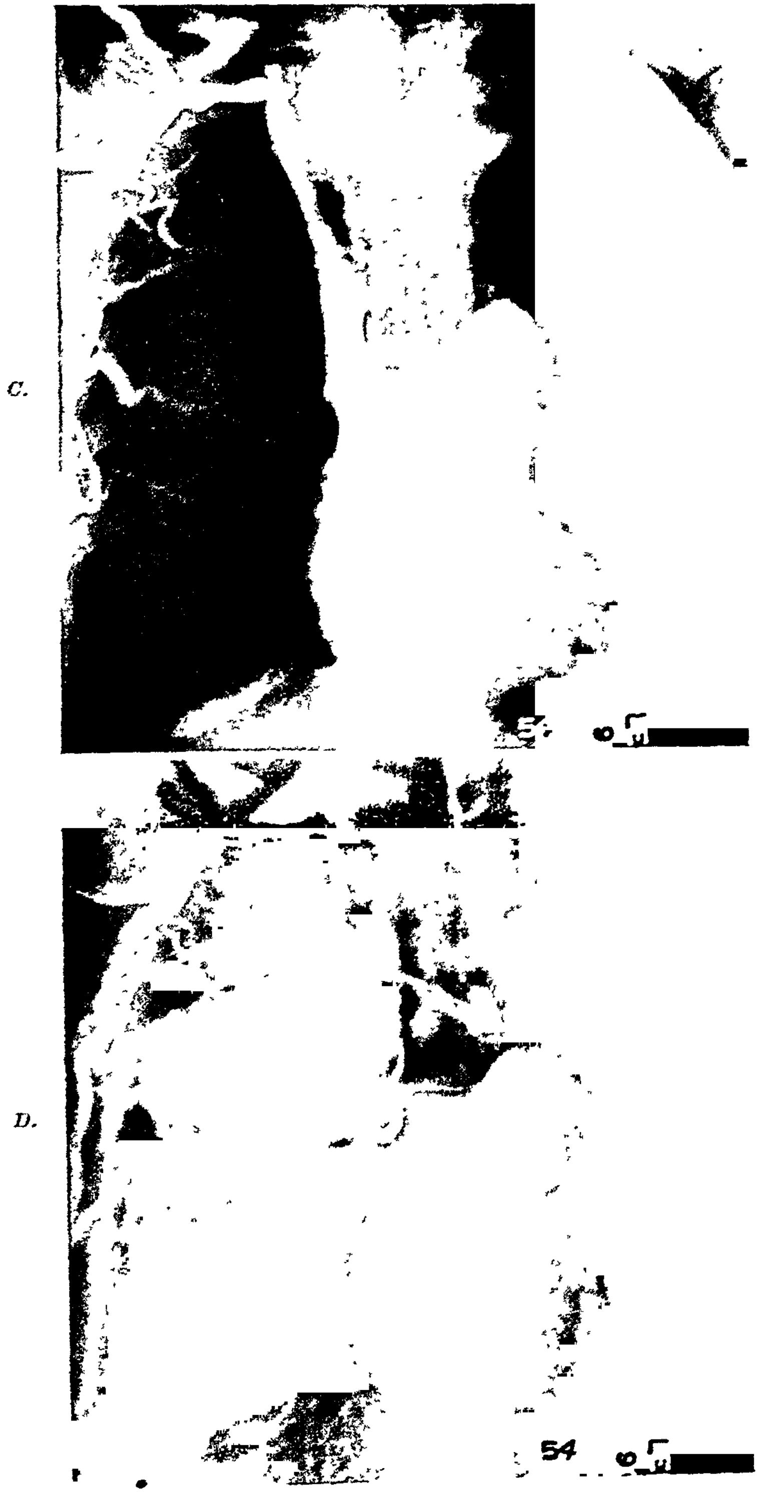


Fig. 4. (Cont'd).—*C*, Frontal angiocardiogram reveals narrowing of the right innominate causing collaterals. Note the markedly narrowed trachea. *D*, Frontal angiocardiogram ater time reveals collaterals entering the vena azygos system.

The size of the mediastinal component alone is often sufficient to contraindicate the attempt to excise it through an exclusively cervical approach. The difficulties in Cases 2, 3, 4, and 5, in which preliminary thoracic excision was necessary, amply support this belief. Furthermore, careful study of the relationships of these tumors to surrounding structures should arouse some suspicion as to the acceptability of blind dissection in the neighborhood of such structures as both pleural spaces, the great vessels, the esophagus, and the trachea. Unless one is prepared to trust to luck in the presence of uncontrolled vascular tributaries, thoracic exposure must be obtained at least in some instances.

In addition to these general objections, our experience further indicates that more careful evaluation, particularly by tomography and angiocardiography, permits classification sufficient to clarify the mode and time of departure from uniform cervical approach.

I. Superior mediastinal goiters, which displace the trachea and esophagus to the left, present predominantly in the right superior mediastinum and displace the right great vessels anteriorly and to the right. Any substernal portion may compress the left innominate vein. These tumors more often displace than concentrically compress the trachea. A combination of cervical and right transthoracic incisions has provided excellent exposure under such circumstances (Cases 1, 2, and 5). With adequate opening of the mediastinal pleura, the vessels have been readily freed and retracted anteriorly. This has permitted, under direct vision, mobilization of the tumor from the lateral aspect of the bronchus and trachea and particularly, as in Case 2, from the esophagus. Manipulation in both the neck and thorax has permitted passing the thoracic portion into the cervical area and then excising it en masse with the right lobe. It also has been possible when necessary, as in Cases 2, 3, and 5, to excise the thoracic portion thus allowing the remainder to be delivered into the neck and removed with the attached thyroid gland. The excellent visualization of the posterior superior mediastinum and the lesser risk of opening both pleural spaces make this approach preferable to the sternal splitting incision which, however, may also be used.

II. Superior mediastinal goiters which displace the trachea and esophagus to the right expand from a position posterior to and on the right of the axis of the aortic arch and the left great vessels. These split the great vessels of the two sides in opposite directions. They may cross the midline. Thus, in Case 3, the left brachiocephalic vessels were displaced to the left while the substernal and right portions of the tumor compressed and displaced the right innominate vein and superior vena cava to the right and anteriorly, but the innominate artery posteriorly. It should be noted that previous substernal thyroidectomy, through an exclusively cervical incision, had been unsuccessful. Such an unusually large and lobulated tumor should be approached between the great vessels of both sides in order to expose both sides of the trachea. Under such circumstances, a combination of low collar and sternal splitting incisions has proved very satisfactory. Careful inspection of the angiocardiography (Fig. 4, *C* and *D*), the right innominate vein was narrowed sufficiently to produce collateral circulation through the azygos system. The pulmonary artery and branches were moderately enlarged and the brachiocephalic vessels of both sides were displaced laterally. Tomography failed to reveal any cartilaginous rings. A primary collar incision, supplemented by a sternal splitting incision, was selected with the hope of avoiding opening the pleural spaces in the presence of tracheal compression requiring tracheotomy.

*Operation.*—On Feb. 5, 1954, the thyroid was exposed through a collar incision. Since removal required further exposure, the sternum was split to the level of the fourth rib. By spreading the sternum and converting both incisions to a large, triangular area, excellent visualization was obtained. After both pleural reflections were mobilized with special care to avoid opening either pleural space, the inferior portion of the tumor was raised from the aorta. Its size and the transverse course of the left innominate vein prevented block excision. The mass then was mobilized from the anterior surface of the trachea and split longitudinally into two parts, carefully avoiding injury to the left innominate vein. Each half was then mobilized from below upward and, after ligation of many vessels, excised separately. The compressed section of trachea did not have its normal cartilaginous support and partial withdrawal of the endotracheal tube was followed by tracheal collapse and inspiratory stridor. The tube was advanced for support while a tracheotomy was performed through which a long endotracheal tube permitted continuation of anesthesia and maintained an excellent postoperative airway for ten days. The cervical portion of the trachea was supported by bilateral suturing to the medial border of the sternocleidomastoid muscles. The wounds were closed, and drainage was employed for twenty-four hours.

*Pathologic Diagnosis.*—Nodular goiter with cystic degeneration and involution.

CASE 5.—The patient, a 61-year-old white man, complained of mild intermittent dysphagia and choking sensations. Distended neck and chest veins and an enlarged nodular thyroid were the significant physical findings. Chest roentgenograms (Fig. 5, *A*) showed a large right superior mediastinal mass displacing the trachea to the left but not seriously compressing it. Tomography showed that this displacement was accompanied by lateral compression and loss of the cartilaginous rings on the side of the tumor, as contrasted with normal rings on the opposite side. Angiocardiography demonstrated abundant collateral circulation due to obstruction of the superior vena cava. A right thoracic incision combined with the low collar incision was selected.

*Operation.*—On Dec. 22, 1955, the right thorax was opened through the bed of the right anterior third rib. The mass extended from the right upper lobe bronchus to the thoracic inlet and displaced the trachea to the left. Through an incision in the mediastinal pleura, it was mobilized from the trachea and esophagus posteriorly after the great vessels had been freed and retracted anteriorly. Despite this mobilization, the tumor could not be delivered into the cervical incision even after division of the strap muscles including the sternal head of the right sternocleidomastoid. Following excision of its major portion within the thorax, delivery and excision in the neck were readily accomplished. The wounds were closed with drainage of the right chest without repair of the mediastinal pleura.

*Pathologic Diagnosis.*—Nodular goiter with marked cystic degeneration.

## DISCUSSION

Mediastinal tumors of thyroid origin are not uniform in size, location, or mode of connection to the parent gland. An accurate knowledge of the relationship of these thoracic goiters to the great vessels, trachea, and the esophagus provides the best basis for the most suitable surgical approach. These cases were specifically selected because they demonstrate great variation in detailed findings which cast doubt on the contention that all can be safely removed through the cervical approach.

## REFERENCES

1. Lahey, F. H., and Swinton, N. W.: Intrathoracic Goitre, Surg., Gynec. & Obst. 59: 627, 1934.
2. Lahey, F. H.: The Surgical Management of Intrathoracic Goitre, Surg., Gynec. & Obst. 53: 346, 1931.
3. Wakely, C. P. G., and Mulvany, J. H.: Intrathoracic Goiter, Surg., Gynec. & Obst. 70: 702, 1940.
4. Sweet, R. H.: Intrathoracic Goiter *Located in the* Postmediastinum, Surg., Gynec. & Obst. 89: 57, 1949.
5. Bressler, S., and Thompson, S. A.: Posterior Mediastinal Goiter, Am. J. Surg. 85: 237, 1953.
6. Ellis, F. H., Good, C. A., and Seybold, W. D.: Intrathoracic Goiter, Ann. Surg. 135: 79, 1952.
7. Rives, J. D.: Mediastinal Aberrant Goiter, Tr. Am. S. A. 65: 417, 1947.
8. Adams, H. D.: Transthoracic Thyroidectomy, J. THORACIC SURG. 19: 741, 1950.

operative details of this case shows that the position of the great vessels over the face of the tumor made right and left thoracic approach undesirable. In contrast to Case 4, both pleural spaces were opened and drained without fear since tracheotomy was not anticipated in this patient. Again, the low collar incision has provided for ready delivery and excision of the remaining portion of the thyroid. Thoracic drainage alone has proved sufficient.

III. Superior mediastinal goiters which surround the trachea often severely compress it in a concentric manner without significant displacement and usually at or just below the thoracic inlet. The serious encroachment on the airway is also complicated by an extensive loss of normal cartilaginous support over the entire circumference in the area involved. Such findings require caution since an incompetent airway may result from removal of the supporting structure of the thyroid gland unless maintained through tracheotomy and other measures as in Case 4. This contrasts with the findings in the other cases in which, although tracheal displacement has been accompanied by some deformity and softening on the side of pressure, the patency has been uniformly maintained by the undisturbed cartilaginous support in the remaining circumference making tracheotomy unnecessary. Tomographic survey of the tracheal rings in the tumor area has been a very satisfactory method of demonstrating such changes. The contrast is well illustrated by comparison of the x-rays of Case 4 with the others, particularly Case 5. The probable necessity for tracheotomy has rendered removal without pleural opening very desirable since a sucking wound with a leaking tracheotomy may carry serious hazard of pleural complications. Under these circumstances, a low collar incision with partial or complete splitting of the sternum has provided satisfactory exposure with maximal control of the airway and pleural problems. If one or both pleural spaces are accidentally opened, both anterior and posterior thoracic tube drainage should be instituted.

## SUMMARY

1. In some patients with intrathoracic tumors of thyroid origin, the additional exposure gained by thoracic incision, supplementing the usual cervical incision, decreases the difficulties and risks of operation through the cervical approach alone. Such a combined approach is particularly useful for extremely large tumors with complex relationships to surrounding vital structures.

2. Tracheotomy is usually unnecessary when the trachea is simply displaced but, with concentric compression and loss of support by the cartilaginous rings, it is an indicated routine. Under such circumstances, exposure should be planned with the hope of avoiding the opening of the pleural spaces.

3. The most suitable approach can be planned with information gained when tomography and angiocardiography are combined with the routine studies in defining the anatomic relationships.

4. Five cases have been reported to illustrate the details of the diagnostic and therapeutic planning.

When this was done, clear fluid was seen to pulsate out of the neck with every heart beat, and the index finger could be passed through the neck into the vertebral canal. The fluid was deduced to be cerebrospinal fluid, although the spinal cord was not visualized The neck of the sac was closed with interrupted No 4 0 silk sutures so that no further leak occurred After the removal of the cyst, the lung was able to be expanded to fill about three quarters of the chest cavity Accordingly, a catheter was inserted into the chest and the chest closed in routine manner The catheter was connected to underwater seal drainage

Three days later the lung had expanded well, but 400 c c of blood tinged fluid was aspirated from the chest Four days after the operation, the patient complained of marked pain down the ventral aspect of both thighs and weakness of the right leg. Straight leg raising of the right leg was limited to 20 degrees by pain in the front of the right thigh Patella and ankle clonus were present on the right There was a right Babinski response There were no sensory changes

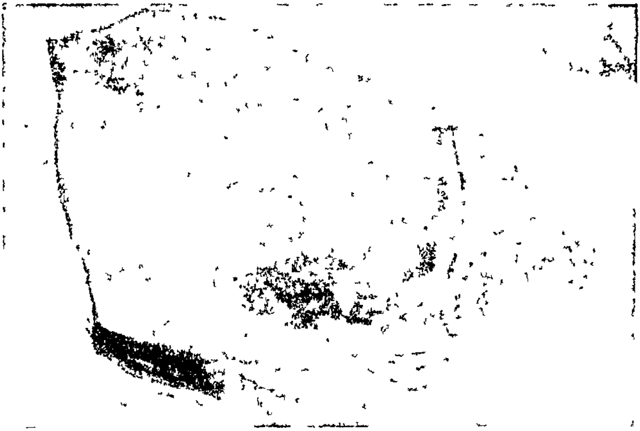


Fig 4 —Appearance of meningocele at operation

Eight days after operation she was seen by Dr Alan Bailey for neurologic assessment and he reported a partial Brown Séquard syndrome with a lesion high in the thoracic area

A lumbar puncture done on the sixth postoperative day gave xanthochromic fluid with 520 mg. of protein per cubic centimeter, so that Dr Bailey suggested that there had been some hemorrhage into the cord at the site of the operative removal of the meningocele.

The patient made a slow but steady recovery with the return to her preoperative condition as regards her neurologic status The ventilatory function studies were repeated seventeen days after operation These showed a definite improvement of ventilatory capacity The lung had fully expanded (Fig 5)

*The Pathological Report* (Dr. D F Moore) —A collapsed saclike structure was seen One pole was open It measured 15 cm in length and had a maximum circumference of 21 cm Adjacent to the opened end, the wall was pale pink in color, averaging 0 2 cm in thickness At the opposite end, the tissue became whitish gray and almost gelatinous in consistency with the thickness increased to 2 6 cm Externally this sac was fairly smooth, internally it had a wrinkled, leathery appearance

X-ray of the lung fields was reported by Dr. E. W. Spencer as follows: "Postero-anterior and lateral views of the chest show the presence of a tremendous mass occupying a greater portion of the right hemi-thorax, and in lateral projection this seems to lie primarily in the middle of the posterior mediastinum." (Fig. 3.) X-ray of the thoracic spine showed a gross kyphoscoliosis of such a degree that, in the anteroposterior projection, a direct view was obtained along the lumen of the upper thoracic vertebral canal. The apex of the kyphosis appeared to be at the level of the fourth thoracic vertebra. No obvious enlargement of the intervertebral foramina could be demonstrated radiologically.

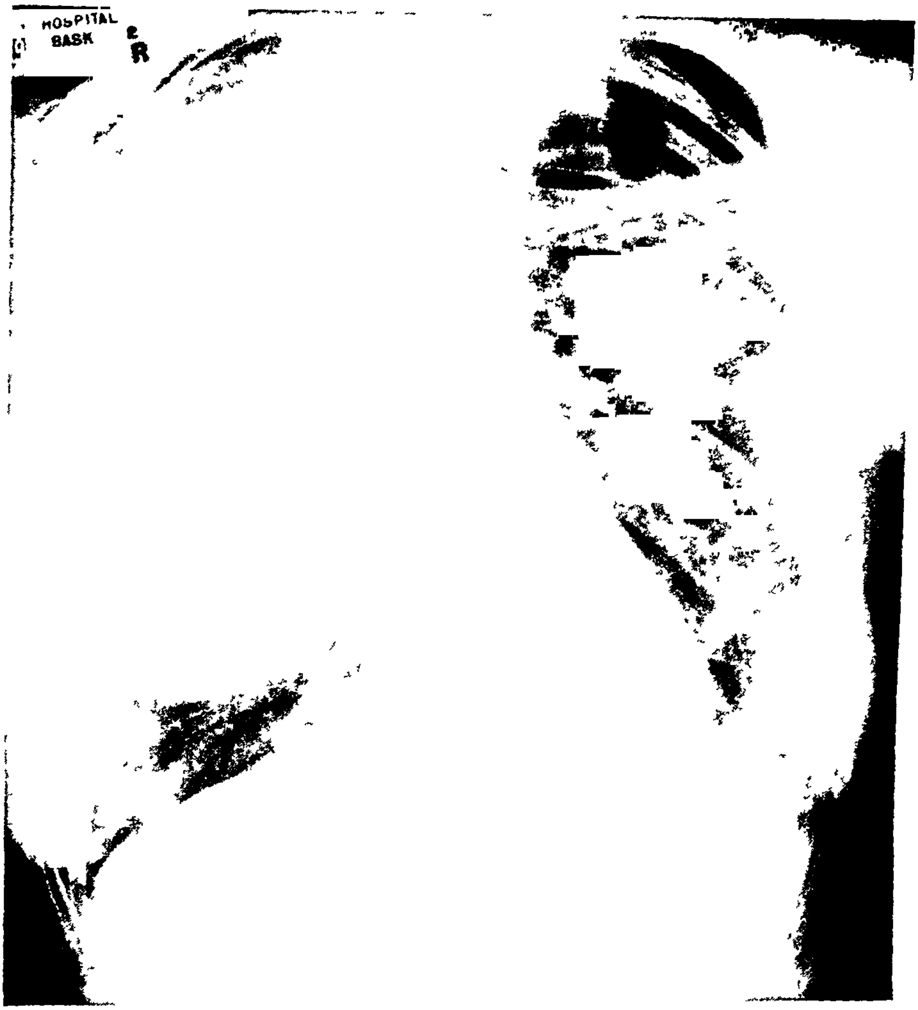


Fig. 3.—Posteroanterior x-ray of chest showing huge meningocele on right side. September, 1955—taken 2 weeks before operation.

Ventilatory function was evaluated by Dr. J. E. Merriman. The vital capacity was 58 per cent of normal. The maximum breathing capacity was 76 per cent of normal.

The preoperative diagnosis was a large neurofibroma occupying the right chest.

Accordingly, on Sept. 8, 1955, I performed a right lateral thoracotomy. The anesthetist was Dr. Gordon M. Wyant.

The right chest was largely occupied by a large, smooth, thick-walled cyst containing clear brown-tinged fluid (Fig. 4). The cyst was attached in the paravertebral gutter in the region of the fourth thoracic vertebra at the apex of the acute kyphosis. The cyst was entered and dissected free to its neck, which was divided at the level of the intervertebral foramen.

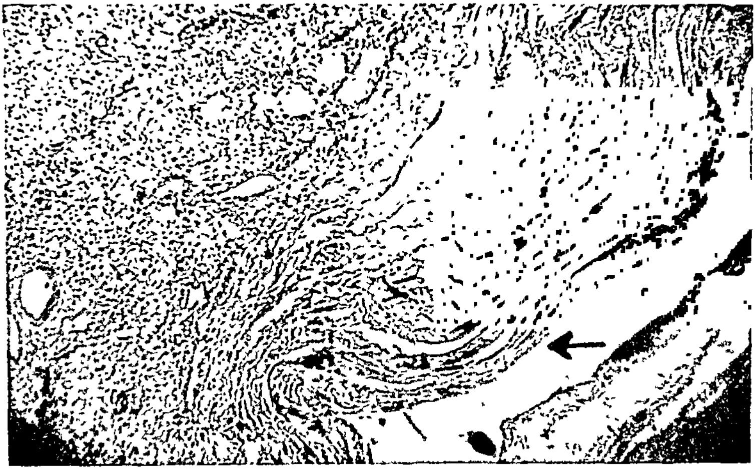


Fig. 6.—Microscopic slide of the wall of thoracic meningocele showing the meninges (arrow) and also the tissue typical of a neurofibroma. (Low power.)

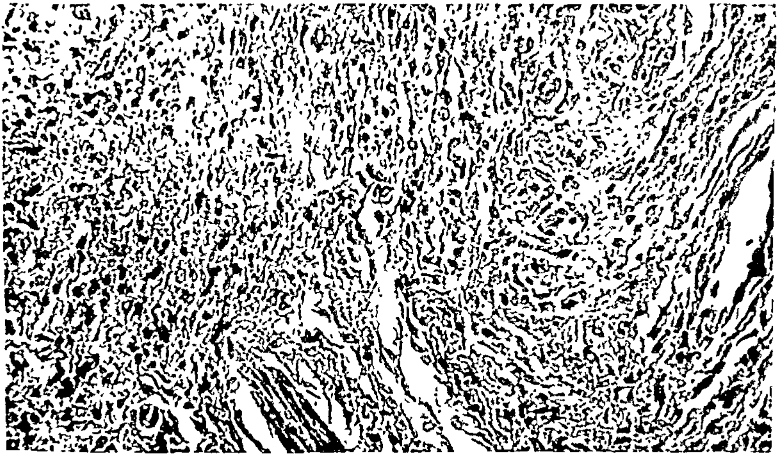


Fig. 7.—Microscopic section of subcutaneous neurofibroma which is exactly similar to the tissue in the wall of the meningocele. (High power.)

*Histologically*, the cavity was lined by pale amorphous eosinophilic material, sometimes laid down in vague lamellae. In the region of the surgical opening, vestiges of leptomeninges were found. These were undergoing bland degeneration and patchy calcification.

The bulk of the edematous wall was composed of loosely arranged, elongated cells with medium-sized fusiform nuclei. These cells seemed identical to those noted in the subcutaneous neurofibroma excised at the same time. This portion was well vascularized. It was encased in fibrotic parietal pleura, overlying peripheral nerve fibers. Near the neck of the sac, a few ganglion cells were incorporated in the nerves (Figs. 6 and 7).

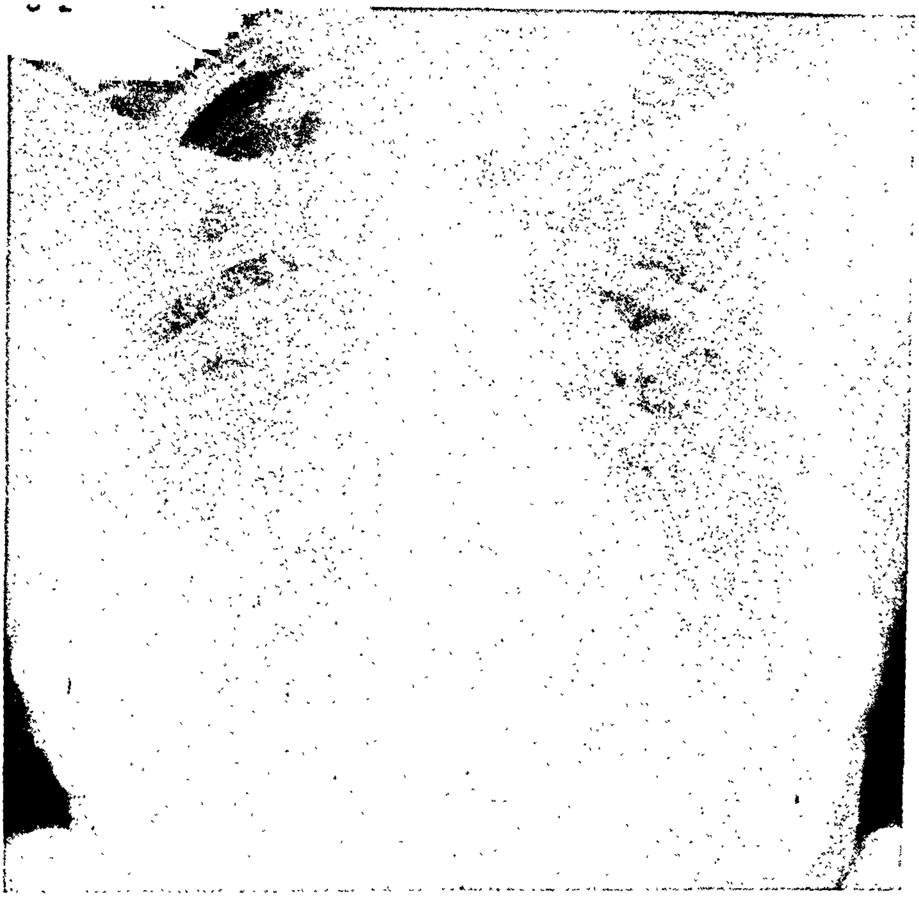


Fig. 5.—Chest x-ray after excision of meningocele showing complete re-expansion of lung.

## DISCUSSION

As in previously reported cases, the diagnosis was not suspected until operation. The excuse for this is that the syndrome was not then known to the author. The patient had suffered from the manifestations of von Recklinghausen's disease since early life. Five years before she was seen at the University Hospital, she had had a tuberculosis survey film of the chest taken and this had showed the mass in the right hemithorax. Then, in 1953, she had fallen on her buttocks and had sustained cord damage. Both she and her mother state that the kyphoscoliosis had not been present before this time, but it is hard to accept this as fact. Chest x-ray at that time showed the mass in the right hemithorax to occupy about two thirds of the right lung field (Fig. 1).

in 1949, reported the case of 22-year-old soldier with neurofibromatosis who suffered a spine injury by being blown out of a tank. Three years later, because of pain, he was investigated and seen to have a kyphoscoliosis at the level $T_8$-$T_{10}$. At this level, there was a meningocele on the right side. The diagnosis was proved by myelography. The treatment was conservative.

Otani,[10] in 1951, made a diagnosis of intrathoracic meningocele in a 40-year-old woman, by means of direct puncture of the cyst. By percutaneous puncture, he aspirated clear fluid which, by analysis, he showed to be exactly similar in composition to the cerebrospinal fluid.

In 1951, Kessel[11] reported 2 cases. The first was a 23-year-old woman with a marked kyphoscoliosis and a meningocele situated at $T_5$-$T_6$, eroding the pedicles of three vertebrae on the left side. It was successfully removed. The second case was in an 8-year-old girl who, at 3 years of age, had an acute mid-dorsal scoliosis. At the age of 7 years she suffered a fall. The scoliosis became worse and the left leg became spastic—then both legs became spastic. She had developed an acute kyphoscoliosis. X-ray showed gross lateral displacement of $T_7$-$T_8$ vertebrae. The meningocele was 3 cm. by 2.5 cm.

Kessel[11] states that up to that time 8 cases were recorded in the literature and his 2 made a total of 10, but of these 10, 7 were associated with von Recklinghausen's disease.

In 1952, Ciaglia[12] reported the case of a 31-year-old woman with neurofibromatosis and acute kyphoscoliosis at $C_7$-$T_1$ level, who had a meningocele at the left apex of the chest which contained 200 c.c. of fluid. It was successfully removed and the patient was well three and one-half years later.

Rubin and Stratemeier[13] record the youngest patient in the series. In 1952, an infant, aged 7 weeks, was admitted to the hospital because of a progressive respiratory distress due to an anterior thoracic meningocele displacing the trachea and esophagus to the left. There were multiple vertebral abnormalities. Unfortunately, the child died before treatment could be instituted and the defect was found to be at the fourth thoracic vertebral level.

In the Swedish literature, in 1952, Turunen[14] reported the case of a man, aged 33 years, with neurofibromatosis and kyphoscoliosis. He had a meningocele, 4 cm. in diameter, at the level of the $T_4$-$T_5$ on the left side. Preoperatively it was thought to be a neurofibroma. It was successfully removed by Nylander.

Cmyral,[15] in 1952, reported the first case of bilateral thoracic meningocele in a man of 53 years of age. This patient had a scoliosis but no mention is made of neurofibromatosis. The diagnosis was proved by injection of Lipiodol into the cyst. No definite treatment was given.

In 1953, Baker and Curtis[16] gave the most comprehensive review of the subject to date and recorded 2 cases. The first was a 40-year-old woman with neurofibromatosis but no spinal deformity. The meningocele was 4 cm. by 5 cm. by 5 cm. and was situated at $T_8$-$T_9$ on the left side. It was diagnosed preoperatively by myelography and successfully excised. The second case was

Some progression of the size of the mass had occurred over the succeeding three years and progressive shortness of breath had occurred. This was relieved by excision of the cyst and re-expansion of the right lung. Following operation some further temporary cord damage occurred.

The danger of removal of these intrathoracic meningoceles has been shown by other writers. Mendelsohn and Kay[1] (1949) go so far as to say that they doubt the advisability of operating on these patients unless their symptoms are progressive.

The first recorded case in the literature is that of Pohl[2] (1933). He operated on a 47-year-old woman with neurofibromatosis, kyphoscoliosis, and a thoracic meningocele on the right side, but the patient died of empyema. In 1938, Schüller and Uiberall[3] recorded the case of a man 56 years of age, who had neurofibromatosis plus a thoracic meningocele, who died following nerve root section of $T_6$-$T_8$ for pain. The meningocele was demonstrated by myelography and confirmed at autopsy.

Ameuille, Wilmoth, and Kudelski[4] recorded a case of a 48-year-old woman, in 1940, in whom the meningocele was situated at the level of $T_8$-$T_9$. She died of empyema following an operation which had been performed because of the preoperative diagnosis of hydatid cyst.

Cabello-Campos[5] (1946) records the cases of 2 patients diagnosed without confirmation. Segnpiel, Ruzicka, and Lodmell,[6] in 1947, reported a thoracic meningocele on the right side in a 21-year-old soldier. Operation was undertaken, but aspiration of the cyst only was done and the diagnosis subsequently confirmed by myelography. This case was subsequently reported again by Byron and his associates,[7] in 1949.

In 1948, Welch, Ettinger, and Hecht[8] recorded the case of a 41-year-old woman with a neurofibromatosis, marked kyphoscoliosis, and a posterior sulcus tumor at the level of $T_6$-$T_7$ with enlargement of the intervertebral foramen. The lesion was a meningocele successfully removed by operation.

Mendelsohn and Kay[1] in 1949, recorded the case of a 40-year-old woman with neurofibromatosis and meningocele, 3 cm. by 4 cm. by 8 cm., in the right chest which communicated with the subarachnoid space through the seventh intervertebral foramen. After excision, a moderate degree of headache and backache persisted for several weeks.

In the same year Byron, Alling, and Samson[7] recorded 2 new cases. A 36-year-old woman, with a known 10-year history of the lesion at the right apex involving the intervertebral foramen between $T_2$-$T_3$ vertebrae. Nine years later, there was a large lesion occupying the right upper chest, causing dyspnea. She had a marked scoliosis. The lesion was successfully removed. The second case was a 61-year-old man with neurofibromatosis and kyphoscoliosis. The cyst was 7 cm. in diameter and occupied the right apex of the chest. The patient died at operation of hemorrhage and cardiac arrest.

In the discussion on Byron's[7] paper, Head spoke of a 48-year-old man who had a meningocele diagnosed by lumbar puncture. Aspiration of the cyst revealed exactly similar fluid samples. Cross, Reavis, and Saunders,[9]

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Otani | 1951 | 1 | 40 | F | No | Yes | 5 cm by 3 cm | $L_5$, Right | Nil | |
| Ciaglia | 1952 | 1 | 31 | F | Yes | Yes | | C, Left | Excision | Recovered 3½ years |
| Rubin and Stratemeier | 1952 | 1 | 7 weeks | ? | ? | | | $T_4$, Left | Died before treatment could begin | Died |
| Turunen | 1952 | 1 | 33 | M | Yes | Yes | 4 cm diameter | $T_5$ $T_6$, Left | Excision | Recovered |
| Cmyral | 1952 | 1 | 53 | M | ? | Yes | ? | Bilateral | Nil | |
| Baker and Curtis | 1953 | 1 | 40 | F | Yes | No | 4 cm by 5 cm by 5 cm | $T_8$ $T_9$, Left | Excision | Recovered |
| | 1953 | 1 | 63 | M | No | No | | $T_9$ $T_{10}$, Right | Excision | Died, Paget's disease, 9 months |
| Sears, Clayton, and Siebel | 1953 | 1 | 29 | F | No | Yes | 10 cm diameter | $T_2$ $T_3$, Left | Excision | Recovered |
| Gernez Rieux and LePaul | 1954 | 1 | 59 | M | No | Yes | 4 cm diameter | $T_{11}$ $T_{12}$, Right | Nil | |
| Hackensellner | 1954 | 1 | 45 | F | Yes | Yes | 5 cm diameter | $T_9$ $T_{10}$ Bilateral | Nil | Died |
| Wilhelm | 1954 | 1 | 38 | F | Yes | Yes | 5 cm | $T_1$, Right | Operation | Died |
| Nanson | 1956 | 1 | 13 | F | Yes | Yes | 15 cm diameter | $T_3$ $T_4$, Right | Excision | Recovered |

TABLE I. CASES OF INTRATHORACIC MENINGOCELE FROM THE LITERATURE

| AUTHOR | DATE | NUMBER OF CASES | AGE (YEARS) | SEX | WITH VON RECKLINGHAUSEN'S DISEASE | KYPHOSCOLIOSIS | SIZE | SITE | TREATMENT | RESULT |
|---|---|---|---|---|---|---|---|---|---|---|
| Pohl | 1933 | 1 | 47 | F | Yes | Yes |  | $T_4$, Right | Excision | Died; empyema |
| Schüller and Uiberall | 1938 | 1 | 56 | M | Yes | Yes |  | $T_6$, Right | Section nerve root, $T_6$-$T_7$ | Died; cardiac arrest |
| Ameuille, Wilmoth, and Kudelski | 1940 | 1 | 48 | F | No | Yes |  | $T_8$-$T_9$, Right | Cyst, packed with gauze | Died; empyema |
| Cabello-Campos | 1946 | 1 | 7 | M | No | Yes | 5 cm. diameter | Upper Thoracic, Right | Nil | Unknown |
|  |  | 1 | 54 | M | Yes | No |  | $T_3$, $T_4$, $T_5$, Right | Unknown | Unknown |
| Freedlander | 1946 | 1 | Middle Age | F | Yes |  |  | ? | Unknown | Unknown |
| Sengpiel, Ruzicka, and Lodmell | 1947 | 1 | 21 | M | No |  |  | $T_1$-$T_2$, Right | Operation, aspiration of cyst | Recovered |
| Welch, Ettinger, and Hecth | 1948 | 1 | 41 | F | Yes | Yes | Neck 3 cm. by 4 cm. | $T_6$-$T_7$, Left | Excision | Recovered |
| Mendelsohn and Kay | 1949 | 1 | 40 | F | Yes | No | 3 cm. by 4 cm. by 8 cm. | $T_6$, $T_7$, $T_8$, Right | Excision | Recovered; headaches |
| Byron, Alling, and Samson | 1949 | 1 | 36 | F | Yes | Yes | 10 cm. diameter | $T_2$-$T_3$, Right | Excision | Recovered |
|  |  | 1 | 61 | M | Yes | Yes | 7 cm. diameter | $C_7$-T , Right | Operation | Died; cardiac arrest |
| Head | 1949 | 1 | 48 | M | ? | ? | ? | ? | Aspiration | Unknown |
| Cross, Reavis, and Saunders | 1949 | 1 | 22 | M | Yes | Yes | 4 cm. by 8 cm. | $T_8$-$T_{10}$, Right | Nil | Unknown |
| Kessel | 1951 | 1<br>1 | 23<br>8 | F<br>F | No<br>Yes | Yes<br>Yes | Small<br>3 cm. by 2.5 cm. | $T_5$-$T_6$, Left<br>$T_7$-$T_8$, ? | Excision<br>Unknown | Recovered<br>Unknown |

a posterior sulcus tumor in this disease is a neurofibroma. Yet in actual fact this association is extremely rare. It would appear that meningocele is a much more common entity.

Furthermore, in considering mediastinal tumors, meningocele is rarely mentioned in the differential diagnosis. Blades,[21] in his review of 109 cases of mediastinal tumors, does not mention it.

TABLE II. SUMMARY OF CASES INTRATHORACIC MENINGOCELE FROM THE LITERATURE

| NUMBER OF CASES | AVERAGE AGE | SEX | WITH VON RECKLINGHAUSEN'S DISEASE | SIDE | TREATMENT | RESULTS | |
|---|---|---|---|---|---|---|---|
| 27 | 40 years | 15 Female | 16—Yes | 15—Right | 16—Operation | Died | 7 |
| | | 11 Male | 8—No | 7—Left | 7—No operation | Recovered | 11 |
| | | 1 Unknown | 3—Unknown | 2—Bilateral | 1—Died before treatment | Unknown | 9 |
| | | | | 3—Unknown | 3—Unknown | | |

The pathology of the 26 recorded cases has not been well defined by the authors. There are very few reports of the exact nature of the wall of the cyst. The present recorded case shows definite evidence that these meningoceles may not arise in the usual manner, but that they may develop from degeneration within a neurofibroma which subsequently communicates with the subarachnoid space. They may commence as a dumbbell type of tumor. The cyst of this case in general had a wall 0.2 cm. in thickness, but at its lower portion it was 2.6 cm. in thickness and in this area it had the structure of a neurofibroma. Furthermore, in one area of the cyst, a myelinated nerve was found.

Kessel[11] suggested that the etiology was to be found in degenerative processes in the bodies of the vertebrae with out-pouching of the meninges. This cannot be substantiated in this case. It is therefore suggested that these meningoceles may indeed commence as true tumors of nerve tissue origin exactly similar to cutaneous lesions and that the subsequent cyst results from degeneration and cavitation with later communication with the cerebrospinal fluid. It is to be hoped that, in future studies of these lesions, very careful attention will be given to the pathologic and histologic picture of the cyst walls.

Surgical excision of the tumor would appear to be the treatment of choice, as the lesion progresses slowly. However, small meningoceles without symptoms could be safely followed conservatively. Modern surgery makes the excision of the cyst relatively safe provided meticulous closure of the neck is carried out.

## CONCLUSION

A case of thoracic meningocele is recorded in association with neurofibromatosis and acute kyphoscoliosis.

a 63-year-old man with no neurofibromatosis and no spinal deformity. The patient had Paget's disease. At operation, a cyst was found at level $T_9$-$T_{10}$ on the right and the diagnosis was established by a type of Queckenstedt's test, with a needle in the cyst. It was successfully excised but the patient died nine months later of Paget's disease.

Sears, Clayton and Siebel,[17] in 1953, record a meningocele in a 29-year-old Negro woman with no evidence of neurofibromatosis. Kyphoscoliosis was present. She had a meningocele, 10 cm. in diameter, situated at level $T_2$-$T_3$ on the left side. It was successfully removed.

Three further examples of intrathoracic meningocele were reported in 1954. Gernez-Rieux, and LePaul[18] describe the case of a 59-year-old man with a meningocele on the right side at the level of $T_{11}$-$T_{12}$. Kyphoscoliosis was present, but no generalized neurofibromatosis. The diagnosis was made by Lipiodol injection. No treatment was given to the patient. In Germany, Hackensellner and Pape[19] report the case of 45-year-old woman who died of a cerebral tumor. She was found to have bilateral meningoceles situated at the level of $D_9$-$D_{10}$. The one on the right side was about the size of an apple and that on the left a little smaller. She had a typical generalized neurofibromatosis. The third case to be reported, in 1954, was that of Wilhelm,[20] who describes a 38-year-old woman who was known to have had a density in the upper right lung field for five years. Four years later, it had enlarged and she had pain in the right shoulder and shortness of breath. She suffered from typical von Recklinghausen's disease and had kyphoscoliosis of the upper dorsal and cervical spine. The meningocele was the size of an apple. She was operated on but died of cardiac arrest and autopsy showed she had a pheochromocytoma as well as an intrathoracic meningocele situated at the level of $T_1$.

This, therefore, brings the list of recorded cases to 27, including the present case. A table (Table I) has been made setting out the characteristics of all these cases and Table II gives the summary of them according to age, sex, association with neurofibromatosis, kyphoscoliosis, and the side of chest involved. The treatment employed and the results are summarized.

It will be noticed that the average age is 40 years. There is a slight preponderance of females over males. At least 60 per cent are associated with neurofibromatosis and kyphoscoliosis. The right side of the chest preponderates over the left. Fifteen patients have been operated on and, of these, 11 recovered.

It would therefore appear that this syndrome has a predilection for middle-aged females and that the right side is more frequently involved than the left.

The diagnosis should be possible before operation by means of myelography and suitable positioning to cause the opaque medium to enter the sac.

It is interesting to note that thoracic meningocele is not generally recorded as a complication of von Recklinghausen's disease. The usual explanation of

# ANTERIOR APPROACH FOR CARCINOMA OF THE SUPERIOR MEDIASTINAL AND CERVICAL SEGMENTS OF THE ESOPHAGUS

WILLIAM R. W[illegible], M.D.,* AND J. GORDON SCANNELL, M.D.**
BOSTON, MASS.

THE surgical ma[illegible] the upper third of the esophagus is difficult becaus[illegible]accessibility of the tumor through the usual thoracotomy [illegible] and because re-establishment of continuity usually requi[illegible] stomach or intestine from the abdomen to the neck. With t[illegible]h, removal of large tumors lying in the thoracic inlet re[illegible] pleural and cervical approach with change of the patie[illegible] operation. To circumvent these difficulties, we prop[illegible]noma of the cervical and superior mediastinal segments [illegible] selected cases through an anterior sternum-splitting ap[illegible] supine throughout the procedure. The distal esophagus [illegible]aneously freed through a laparotomy incision and [illegible]d by gastropharyngeal or high gastroesophageal ana[illegible]ng advanced through the anterior mediastinum. Ideal[illegible]arried out extrapleurally. The application of this a[illegible] high esophageal lesions in the first 6 patients in wh[illegible] the substance of this report.

*Operative Metho*[illegible]s carried out with the patient in the dorsal decubit[illegible]st wall, and abdomen are prepared and draped. T[illegible]st are also prepared so that lateral extensions of t[illegible] can be made, should they be necessary. Initially, [illegible]posed. The skin is incised as for a thyroidectomy [illegible] end of th[illegible]ncision extended up anterior to the sternocleidomas[illegible] almost to [illegible] mastoid process. Dissection is then carried in fron[illegible]uscle, over [illegible]rotid sheath and behind the thyroid to expose the [illegible]phagus ar[illegible]ynx. Lesions in this region can be inspected and [illegible] along with [illegible] lymph nodes. If the lesion is resectable, the skin ov[illegible] sternum is incis[illegible]pes applied, and the sternum split using a Lebsche kn[illegible]eparation of th[illegible] halves of the sternum is accomplished with a smal[illegible] spreader. This [illegible] the upper mediastinum. The pleura is pushed back and the left innominate vein interrupted. Further dissection posteriorly along the left side of the trachea exposes the esophagus

---

From the Surgical S[illegible] Massachusetts General Hospital and the Department of Surgery, Harvard Medical School, Boston, Mass.

Received for publication May 4, 1956.

*Clinical Associate in Surgery, Harvard Medical School; Assistant Surgeon, Massachusetts General Hospital, Boston, Mass.

**Clinical Associate in Surgery, Harvard Medical School; Associate Visiting Surgeon, Massachusetts General Hospital, Boston, Mass.

This would appear to constitute a syndrome which is not generally recorded in connection with von Recklinghausen's disease.

The condition should be capable of diagnosis preoperatively by means of myelography.

The treatment is excision with careful closure of the neck of the sac.

## REFERENCES

1. Mendelsohn, H. J., and Kay, E. B.: Intrathoracic Meningocele, J. THORACIC SURG. 18: 124-128, 1949.
2. Pohl, R.: Meningocele im Brustraum unter dem Bilde eines intrathorakalen, Rundschattensm Röntgenpraxis 5: 747-749, 1933.
3. Schüller, A., and Uiberall, H.: Case of Neurofibromatosis Recklinghausen Combined With Lateral Spinal Meningocele, Confinia neurol. 1: 312-317, 1938.
4. Ameuille, P., Wilmoth, P., and Kudelski, C.: Meningocele rachidienne à developpement intrapleural, Bull. et mém. Soc. méd. de Paris 56: 608-616, 1940.
5. Cabello-Campos, J. M. C.: Meningocele Intratoracico, Bol. San São Lucas 8: 40-48, 1946.
6. Sengpiel, G. W., Ruzicka, F. C., and Lodmell, E. A.: Lateral Intrathoracic Meningocele, *Radiology 50: 515-520, 1948.*
7. Byron, F. X., Alling, E. E., and Samson, P. C.: Intrathoracic Meningocele, J. THORACIC SURG. 18: 294-303, 1949.
8. Welch, S., Ettinger, A., and Hecht, P. L.: Recklinghausen's Neurofibromatosis Associated With Intrathoracic Meningocele, New England J. Med. 238: 622-625, 1948.
9. Cross, G. O., Reavis, J. R., and Saunders, W. W.: Lateral Intrathoracic Meningocele, J. Neurosurg. 6: 423-432, 1949.
10. Otani, G.: Meningocele Intratoracico, Ann. radiol. Diag. (Bologna) 23: 416-419, 1951.
11. Kessel, A. W.: Intrathoracic Meningocele, Spinal Deformity and Multiple Neurofibromatosis, J. Bone & Joint Surg. 33: 92-93, 1951.
12. Ciaglia, P.: Intrathoracic Meningocele, J. THORACIC SURG. 23: 283-292, 1952.
13. Rubin, S., and Stratemeier, E. H.: Intrathoracic Meningocele, Radiology 58: 552-555, 1952.
14. Turunen, M.: Intrathoracic Meningocele, Acta chir. scandinav. 106: 299-303, 1953.
15. Cmyral, L.: Über einen Fall von intrathorakalen bilateraler Meningocele, Radiology Austricia 5: 23-32, 1952.
16. Baker, J. M., and Curtis, G. M.: Intrathoracic Meningocele, West. J. Surg. 61: 216, 1953.
17. Sears, A. D., Clayton, R. S., and Siebel, E.: Intrathoracic Meningocele Not Associated With Neurofibromatosis, J. THORACIC SURG. 26: 101-104, 1953.
18. Gernez-Rieux, Ch., and LePaul, G.: Les meningoceles à developpement intrathoracique, J. franç. méd. et chir. thorac. 8: 633-653, 1954.
19. Hackensellner, H. A., and Pape, R.: Euber Meningkelen Bei Neurofibromatosis Recklinghausen, Fortschr. Geb. Röntgenstrahlen 81: 66-71, 1954.
20. Wilhelm, E.: Meningocele Des Brustraumes, Thoraxchirurgie 2: 147-155, 1954.
21. Blades, D.: Mediastinal Tumours, Ann. Surg. 123: 749-765, 1946.
22. Freedlander, S. O.: Quoted by Mendelsohn and Kay.[1]

down to the level of the aortic arch. In dealing with malignant lesions, the left recurrent laryngeal nerve is routinely sacrificed. Mobilization of the supra-aortic portion of the esophagus is easily carried out under direct vision and separation of the esophagus or tumors from the trachea may be safely carried out. If necessary, portions of the membranous trachea can be excised. In one case, here reported, the tracheal rings were flexible and allowed linear suture of such a defect with only moderate narrowing of the trachea. The esophageal arteries arising from the aortic arch and from bronchial arteries can be secured and ligated under direct vision. Access to the upper mid-esophagus can be facilitated by gently lifting the aortic arch forward and to the left.

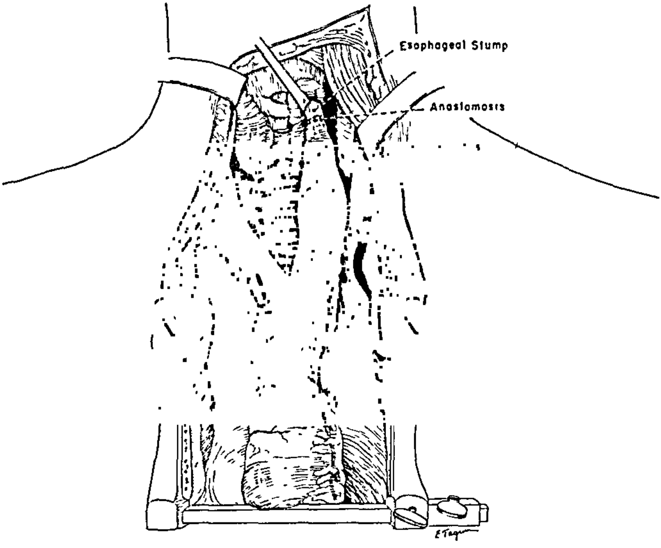


Fig. 3.—The stomach passing through the anterior mediastinum and anastomosed to the uppermost portion of the esophagus after esophagectomy.

In this manner, the normal portion of the esophagus can be freed down to the level of the carina with relative ease. Beyond this, the esophagus is freed by finger dissection. Although occasionally individual vessels can be clamped and ligated, it may be necessary to interrupt several small vessels without ligature. We have seen only minimal bleeding from such a maneuver.

As soon as the resectability of the lesion is established, a second team begins mobilization of the entire stomach through a midline upper abdominal incision. The freeing of the stomach is carried out so as to preserve the right gastric and right gastroepiploic vessels. The short gast nd le stro-epiploic vessels are divided. The left gastric artery is ca in

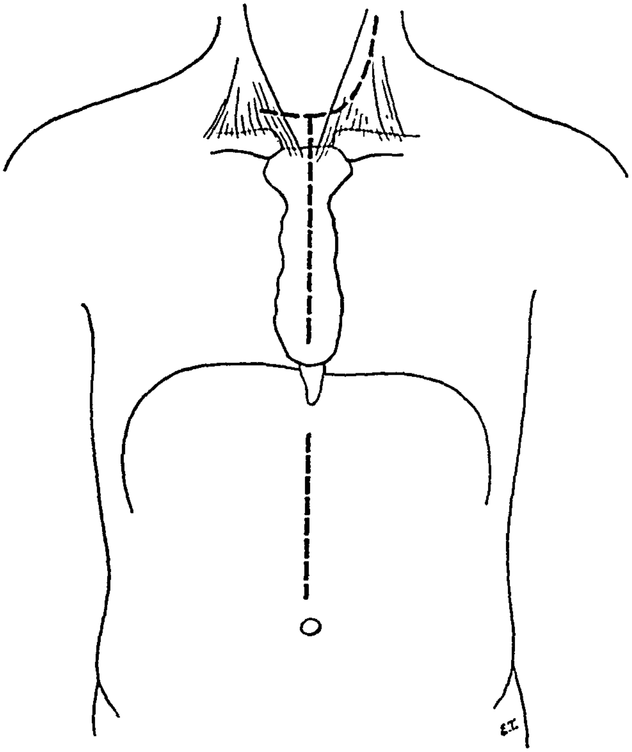


Fig. 1.—The skin incisions used for cervical, mediastinal, and abdominal approach to esophagus and stomach.

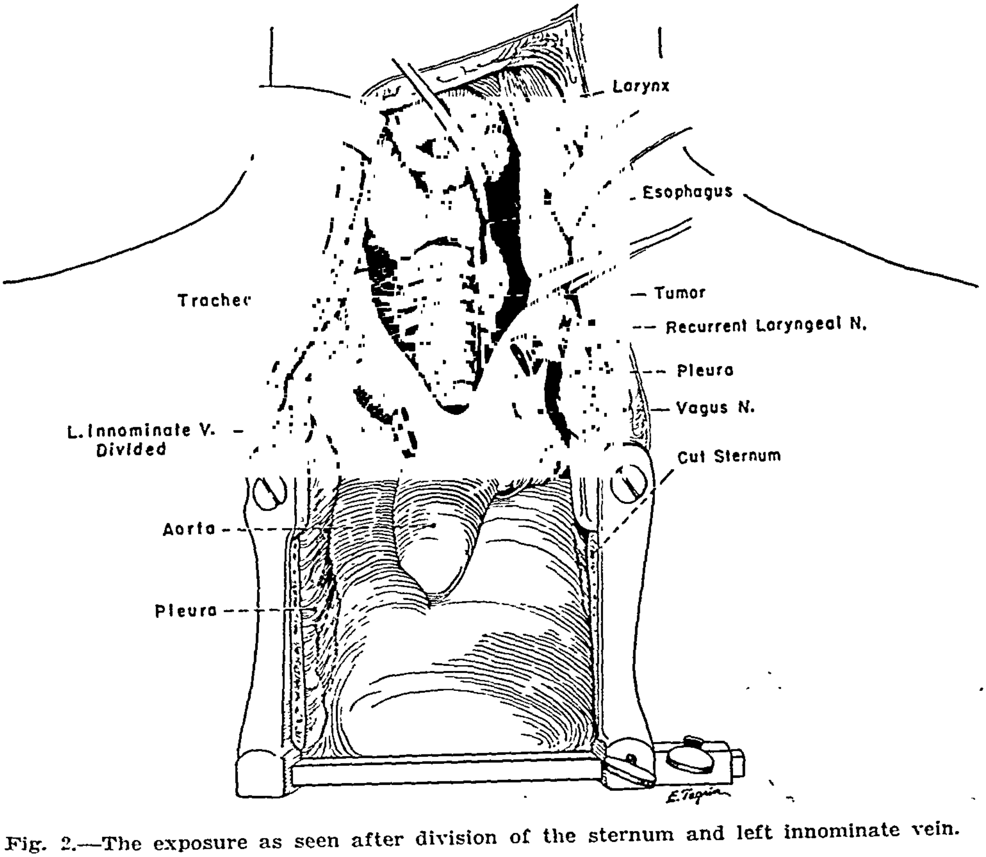


Fig. 2.—The exposure as seen after division of the sternum and left innominate vein.

On May 13, 1955, a total esophagectomy was carried out through cervicomediastinal and abdominal incisions. Gastrointestinal continuity was re-established by pharyngogastrostomy—the stomach being advanced through the anterior mediastinum. The tumor invaded the membranous portion of the trachea, a 2 cm. segment of which was removed. The tracheal rings were pliable and it was possible to make a linear closure with only moderate narrowing, posteriorly. One lymph node, of eight examined, contained tumor. The postoperative convalescence was uncomplicated. The patient was discharged on the fifteenth postoperative day.

Approximately six weeks after discharge, a course of radiation amounting to 6,000 r (*tumor dose*) was *given over a period of one month. Shortly thereafter he returned to work.* He has remained well during the ensuing year.

CASE 3.—M. G. H. (No. 894762), a 58-year-old truck driver, entered the hospital complaining of dysphagia of two months duration. The esophagus was completely obstructed and he had lost 21 pounds of body weight. Except for weight loss there was no physical abnormality. Roentgenographic examination showed an extensive lesion consistent with carcinoma which extended from the hypopharynx to below the aortic arch. Bronchoscopy showed displacement of the trachea but no actual invasion of tumor through the mucosa.

On June 9, 1955, an exploratory operation was performed through a cervicomediastinal incision. The trachea was invaded extensively and there were many grossly involved lymph nodes. The lesion was not resectable. A tracheostomy was established.

The postoperative convalescence was uncomplicated. The patient subsequently died of pneumonia during radiation treatment, presumably having developed a tracheoesophageal fistula as a result of necrosis of tumor.

CASE 4.—M. G. H. (No. 911324), a 56-year-old housewife, came to the hospital because of a 20 pound weight loss and dysphagia. She could eat soft foods with difficulty. Physical examination and all laboratory examinations were normal. Radiographic examination of the esophagus showed an extensive filling defect extending from the cervical region to below the aortic arch. Bronchoscopy showed no involvement of the trachea.

On Nov. 21, 1955, esophagectomy and cervical esophagogastrostomy were carried out through cervicomediastinal and abdominal incisions. During this procedure, the azygos vein was accidentally divided. It was necessary to open the right pleural cavity to control bleeding. The tumor was rather extensive, measuring 8 cm. in length. There were metastases to two paratracheal lymph nodes and three of fourteen paraesophageal nodes, all adjacent to the tumor. In this patient an attempt was made to avoid a tracheostomy. However, this was necessary four days later because she had difficulty raising secretions. On the sixteenth postoperative day, a cervical abscess with a fistulous communication to the esophagogastric anastomotic line was drained.

The fistula gradually closed over the next month, and a course of radiation (3,000 r tumor dose) was given to the upper mediastinum and cervical area. Slow convalescence has continued to date.

CASE 5.—M. G. H. (No. 916654), a 65-year-old carpenter, came to the hospital complaining of dysphagia of three months' duration. Despite a 30 pound weight loss he was still obese. The heart was enlarged, and was fibrillating at a slow rate. He had been taking digitalis for several years. Roentgenographic examination of the esophagus with barium showed a large filling defect involving the upper third of the esophagus. Other laboratory and special diagnostic studies, including bronchoscopy, were normal.

On Feb. 3, 1956, a total esophagectomy with pharyngogastrostomy was performed. The carcinoma of the esophagus extended for a distance of 6 cm., the upper edge being at the cricopharyngeus muscle. There were two lymph nodes involved by tumor. Two days following operation, an embolus lodged in the left brachial artery. An embolectomy was performed with restoration of normal circulation to the extremity. Convalescence was then uneventful until the tenth postoperative day when the patient suddenly died. Autopsy showed a pulmonary embolus to be the cause of death. The mediastinum and the pharyngogastric anastomosis were well healed.

to preserve continuity between right and left gastric vessels along the lesser curvature. The surgeon working from the abdominal approach mobilizes the lower esophagus and draws it down much as in the performance of a transabdominal vagotomy. Through the hiatus, the lower esophageal vessels can be pulled down with a finger and the proximal portions clamped and ligated. Occasionally, a vessel cannot be reached with instruments. If so, it is simply stripped out of the esophageal wall and allowed to retract. In this manner, the dissection from above meets the dissection from below. The entire esophagus is thus freed and, after severance from the stomach, delivered into the superior mediastinum. The mobilized stomach is brought through an opening in the anterior portion of the diaphragm into the anterior mediastinum. The stomach will reach the pharynx by this route without undue tension. A three-layer anastomosis between the stomach and pharynx, or esophageal stump, is made. For this, fine interrupted silk sutures are used.

The abdominal wound is closed in layers using through-and-through stay sutures. The sternum is wired with heavy steel wire.

It is important to establish a tracheostomy before withdrawal of the endotracheal tube. This can be placed to the right of the midline so that the tracheostomy does not communicate with the area of mediastinal dissection.

## CASE REPORTS

Case 1.—M. G. H. (No. 866878), a 62-year-old man, was transferred to the Massachusetts General Hospital from a state tuberculosis sanatorium because of esophageal obstruction of two weeks' duration. He had been treated for extensive fibrocaseous pulmonary tuberculosis for eight years. For five years, he had been paraplegic as a result of cord compression due to tuberculosis of the spine. Roentgenograms demonstrated a filling defect in the cervical esophagus extending down 7 cm., from a point 3 cm. below the cricopharyngeus muscle. There was also extensive fibrosis of the upper lung fields. Bronchoscopy showed a paralyzed left vocal cord and some anterior displacement of the trachea without invasion.

On April 7, 1955, a total esophagectomy and pharyngogastrostomy was carried out through cervicomediastinal and abdominal incisions. A tracheostomy *was not* established.

Postoperatively, he had copious tracheobronchial secretion but coughed effectively until, on the eighth day after operation, the sternal wound separated. About the same time, orally ingested liquids appeared from the mediastinum. A tracheostomy and a feeding jejunostomy were made. Despite these measures he gradually deteriorated and died on the twenty-eighth postoperative day.

Post-mortem examination revealed a tiny leak at the gastropharyngal suture line, dehiscence of the sternal wound resulting from cutting through the bone by wire sutures, localized infection of anterior mediastinum (anterior to transplanted stomach) in continuity with the open sternal incision, pulmonary congestion and edema, pneumonitis, severe tracheobronchitis, fibrocaseous pulmonary tuberculosis, tuberculosis of the spine, chronic cystitis with calculi, pyelonephritis acute and chronic, and atrophy of the spinal cord distal to compression at the collapsed eleventh dorsal vertebra.

Case 2.—M. G. H. (No. 891317), a 70-year-old doorman, entered the hospital with progressive dysphagia of four months' duration. At the time of entry he could swallow only thin liquids. There had been a 20-pound weight loss. Physical examination and all laboratory tests were normal. Radiologic examination with barium showed a large obstructing lesion extending down 4.5 cm. from the level of the sternal notch.

On the basis of our limited experience with this procedure, it appears that a logical modification might be to transect the esophagus beneath the tumor, turn in the distal cut end, resect the tumor and adjacent lymph node areas and restore continuity by transplantation of the colon or jejunum through the anterior mediastinum In most patients, mobilization of the colon is more readily accomplished than freeing of the stomach Particularly in obese and thick-chested individuals, the transabdominal exposure that permits dissection to mobilize the stomach is difficult to obtain Complete longitudinal division of the sternum aids in exposure since the costal margins can be separated once the sternum is split, but even with this aid to exposure this portion of the operation may be difficult and time consuming

The postoperative care of these patients requires that certain precautions be taken A tracheostomy must be performed The transplanted stomach continues to secrete and after the patient begins to take liquids there may be gastric retention The stomach becomes subject to the same fluctuations in pressure as other intrathoracic viscera and especially during coughing there may be regurgitation into the pharynx and spill over into the trachea Also the dissection about the pharynx causes temporary disturbance of swallowing mechanisms There may be aspiration of variable amounts of fluid and food for days, or even weeks, after operation A tracheostomy is necessary to deal with this complication After transplantation of the stomach into the thorax a Levin tube should be left in place and aspirated continuously, until the patient can be upright while taking fluids and until satisfactory emptying of the stomach has been demonstrated

## SUMMARY

A technique for performing total esophagectomy through cervical, trans sternal, and abdominal incisions is described This method has been used for the removal of cervical and superior mediastinal carcinoma of the esophagus Gastrointestinal continuity has been re established by gastropharyngostomy or high gastroesophagostomy, the stomach being advanced through the anterior mediastinum

We are indebted to Dr Edward D Churchill for pointing out the merits of a cervico thoracic approach to a variety of lesions that arise in the superior mediastinum and upper thorax

CASE 6.—M. G. H. (No. 920032), a 60-year-old man, entered the hospital with complete esophageal obstruction. He was emaciated and an indefinite tumor could be palpated in the base of the neck. Roentgenograms showed an extensive ulceration of the esophagus from the pharynx to below the aortic arch. The larynx was invaded and the left vocal cord was paralyzed. A bronchoscope could not be inserted.

On March 22, 1956, the neck and mediastinum were explored. An extensive carcinoma of the esophagus which invaded the larynx and the entire trachea was found. A tracheostomy and a gastrostomy were made. The immediate postoperative convalescence was uneventful. Radiation was begun but the patient deteriorated rapidly and died of bronchopneumonia.

## DISCUSSION

There are several advantages of this method of dealing with lesions of the upper esophagus. It allows easy and direct exposure of the esophagus from the aortic arch to the pharynx. This is possible because of the slight deviation of the esophagus to the left and the ease with which the trachea and esophagus can be rotated to allow dissection. With tumors in the thoracic outlet, the entire dissection of the tumor can be carried out with a single exposure. This avoids partial dissection of the lower end of the tumor from within the chest and the upper end of the lesion from the neck. Once the sternum has been split and the left innominate vein divided, the exposure of the mediastinum is adequate. One of the impressive features of this exposure is the shallow depth between the anterior bony thorax and the anterior surface of the vertebral bodies in the neck and upper thorax. Lower in the thorax, the anteroposterior diameter is greater and exposure of the esophagus more difficult due to the posterior inclination of the spine as it curves dorsally. Nevertheless, dissection is straightforward down to the level of the left main-stem bronchus.

The second advantage of this method of performing total esophagectomy is that neither pleural cavity needs to be entered. Since most of the patients are in older age groups, this feature may allow operation in some patients that otherwise would be considered unable to withstand operation. The pleura was entered in only one of the 6 patients on whom this procedure has been carried out.

The third possible advantage of this approach to lesions of the upper esophagus is that gross extensions of tumor to lymph nodes in both the cervical and upper mediastinal areas can readily be removed with the primary tumor. In each of the 4 patients on whom resections were performed, there were tumor-bearing lymph nodes adjacent to the main tumor and in the sulcus between the trachea and esophagus. In 2 patients, the left lobe of the thyroid was adherent to cervical tumors and was removed in continuity.

The disadvantages of this procedure are several. If one utilizes the stomach for restoration of continuity, the entire distal esophagus must be removed. This involves more or less blind dissection of the esophagus from the level of the carina down to the diaphragm. Although significant bleeding from esophageal arteries stripped from the esophagus wall has not been encountered, this is an inherent danger that might require a radical extension of the approach. In one case, the azygos vein was inadvertently divided and it was necessary to extend the incision into the right pleural space to control bleeding.

replaced with a homograft. If the dissection had involved the arch or ascending aorta, "A window was cut in the intima above and the remainder of the wall was then sutured to obliterate the false passage. Distally, the dissected layers were approximated by a continuous circular suture and the ends of the aorta anastomosed."[2]

These operative procedures attempt to produce a "healed aneurysm," and offer an opportunity for the cure of this otherwise almost hopeless disease. It is worth while to point out that the extent of the dissection and the origin of the initial split in the intima will necessitate modifications of the application of the surgical concept of creation of a "healed aneurysm."

Shennan,[9] who catalogued the largest series of dissecting aneurysms, listed in great detail the extent of the dissection in 297 cases. In 121 instances, the dissection remained limited to the ascending aorta or arch. In 104 cases, it started in the ascending aorta or arch and extended for a variable distance into the descending aorta and sometimes beyond the iliac arteries. In only 62 individuals, did it start at the origin of the left subclavian artery or within the descending aorta so that the arch was entirely free of the dissection. In 10 cases, the dissection started in the abdominal aorta (below the level of the diaphragm).

This is a very clear demonstration of the necessity for modifications that will enable one successfully to attack all such possible problems.

Arterial homografts or substitutions will be most useful in caring for those cases where the dissection has begun beyond the arch or in the abdominal aorta. The full concept of the "healed aneurysm" will be applicable where the dissection has started in the ascending aorta or arch and progressed into the descending aorta or beyond. This still leaves the very large group of patients in whom dissection is only within the arch or ascending aorta and for which no operative procedure has been suggested. Transection of the ascending aorta or arch with present-day methods would seem to be impractical.

The following is the report of an operative case with survival in which a "double-barreled" aorta was created. During the operative procedure, a possible method of treating those patients in whom the dissection is limited to the ascending aorta or arch was suggested.

### CASE REPORT

The patient, (F. W.) hospital No. 55-10107, a 64-year-old white man, was in good health until Sept. 2, 1955. While presiding at a meeting, he developed severe interscapular pain which did not radiate and accompanied by profuse sweating over his entire body. There was no precordial pain. He was hospitalized in his community hospital. One-half grain of morphine did not control the pain, but an additional 150 mg. of Demerol alleviated it. His past medical history revealed that he had been treated for moderate hypertension of 150-160/100 mm. Hg for ten years. At the time of the discovery of his hypertension, a positive serology was noted which was treated with bismuth and arsenicals. The serology remained positive.

Physical examination showed an obese white man in acute distress who presented a shocklike picture with cold clammy sweat over his entire body and a marked pallor. Blood pressure was 160/100 mm. Hg. Examination of the heart [illegible] rhythm with occasional ventricular extrasystoles; no murmurs [illegible] Reflexes were equal and within normal limits in both lower

# SURGICAL TREATMENT OF DISSECTING ANEURYSM

RICHARD A. GILMAN, M.D.,* AND CHARLES P. BAILEY, M.D.**
PHILADELPHIA, PA.

DISSECTING aneurysm has been recognized as a clinical diagnostic and pathologic entity for many years. The disease itself has been described extensively in the literature. Classic among these are the pathologic works of Erdheim[3, 4] and Moritz,[7] the very minute clinical and pathologic study of Shennan,[9] and the clinical review of Levinson, Edmeades, and Griffith.[6]

In spite of all the clinical and pathologic work which has been done, a review of the world literature reveals that proposals for treatment of this condition have been sporadic and scant.

The natural history of the course of dissecting aneurysm suggests a rational approach to the surgical therapy. Once dissection has been initiated there are two avenues of termination. The first is ultimate rupture through the adventitia with hemorrhage and death. The second is a final rupture back through the intima into the aortic lumen with the formation of a "double-barreled" aorta. This represents the so-called "healed aneurysm" which is compatible with life for an indefinite period, but which occurs only in approximately 10 per cent of cases.[10]

Gurin, Bulmer, and Derby,[5] in 1935, were the first to attack a dissecting aneurysm surgically. While operating upon a patient to relieve obstruction to the iliac artery caused by a dissecting aneurysm, they opened the intima allowing the blood in the channel of dissection to return to its normal passage. The patient died on the sixth postoperative day. The case, however, initiated the concept of the surgically created "healed aneurysm." It was not until 1955 that Shaw[8] reported the second attempt. He not only fenestrated the aorta, this time in the abdominal portion, but sutured the dissected layers together distally. This was a second important step as it prevented further dissection beyond the re-entrance point. This patient died on the ninth postoperative day. DeBakey, Cooley, and Creech,[1, 2] in 1955, reported 6 cases treated by surgery with four survivals. Their techniques varied, but basically consisted of transection of the descending aorta with suture of the cut end of the distal segment of the split media and intima of the inner tube to the media and adventitia of the outer, thus obliterating the entrance of blood into the external passage distally. When it was feasible to resect the dissected proximal portion of the aorta entirely, it was

---

This work was supported by the Mary Bailey Foundation for Heart and Great Vessel Research.

Received for publication May 4, 1956.

*Resident Physician, Department of Thoracic Surgery, Hahnemann Medical College and Hospital of Philadelphia, Pa., National Heart Institute Trainee.

**Professor and Head of Department of Thoracic Surgery, Hahnemann Medical College and Hospital of Philadelphia, Pa.

The patient was prepared and taken to surgery on Sept. 9, 1955.

*Operative report:* A standard left posterolateral thoracotomy incision was made, widely opening both the fourth and seventh intercostal spaces to gain complete access to the entire thoracic aorta. Inspection revealed that the proximal portion of the aortic arch was only slightly dilated while the distal arch and descending aorta were dilated to approximately three times their normal size. The adventitia had an ecchymotic appearance. It had been planned to anastomose a whole homologous aortic arch graft obliquely end-to-side to the proximal arch, to divide the lower thoracic aorta, to repair the split coats of the distal segment and to establish continuity by an end to end anastomosis between the graft and the lower segment. Later, the upper dissected segment would have been excised. After application of the modified Satinsky clamp to the ascending aorta, the excluded portion was opened. Two lumens were found in what had appeared to be a normal aorta. Feeling that our previous plan was illogical, the opening was closed and the thought of substitution of a graft for the diseased segment of aorta was abandoned. The opening in the ascending aorta was repaired.

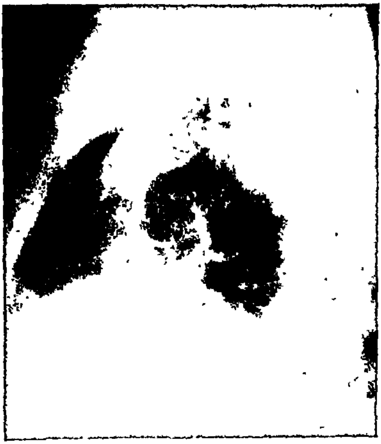


Fig. 2.—Aorta outlined with 70 per cent Diodrast. A double contrast shadow is seen beginning at the origin of the innominate artery and extending parallel with the left half of the aortic arch and the descending aorta.

A modification of the method of DeBakey, Cooley, and Creech was chosen instead. The descending aorta was dissected free, two pairs of intercostal arteries being divided. The aorta was doubly cross-clamped at widely separated points. The combined adventitia and outer portion of the split media were incised transversely into the false lumen anteriorly and on both sides, leaving the posterior half of its continuity intact. This prevented separation of the two ends, facilitating and expediting the final anastomosis. The dissection was found to encompass the entire wall, creating a true double barreled tube. A great deal of soft, jellylike, black clot was evacuated from the false lumen (Fig. 3). The inner tube, consisting of split media and intima, was divided in its entire circumference (Fig. 4). The cut end of the distal split wall, was repaired using a continuous arterial suture of No. 4-0 silk, obliterating the false lumen at this level (Fig. 5). The proximal cut end of the external tube, consisting of the adventitia and the outer portion of the split media, was sutured to the entire thickness of the reconstituted wall of the distal segment by an over-and-over suture of No. 4-0 arterial silk (Fig. 6). This was accomplished with ease, and gave a

The pain was ameliorated with Demerol; severe paroxysms of cough were controlled with Dilaudid. These coughing episodes were accompanied by cyanosis which necessitated continuous oxygen therapy. On Sept. 3, 1955, he developed moist râles throughout both lung fields. X-ray of the chest was interpreted as showing a diffuse dilatation of the ascending aorta, arch, and descending aorta (Fig. 1). A diagnosis of a syphilitic aneurysm of the aorta was made and the patient was transferred to the Thoracic Surgical Service of the Hahnemann Hospital, in Philadelphia, on Sept. 4, 1955. Here a clinical diagnosis of dissecting aneurysm of the thoracic aorta was made.

Upon admission, he was still complaining of pain in the interscapular area and, in addition, a diffuse type of abdominal pain. Blood pressure was 170/80 mm. Hg. There were expiratory wheezes and moist râles throughout both lung fields. The heart revealed a normal sinus rhythm with no murmurs. The abdomen was negative with no tenderness or spasm on palpation. Reflexes were equal and active, and good arterial pulsations were present in the lower extremities.

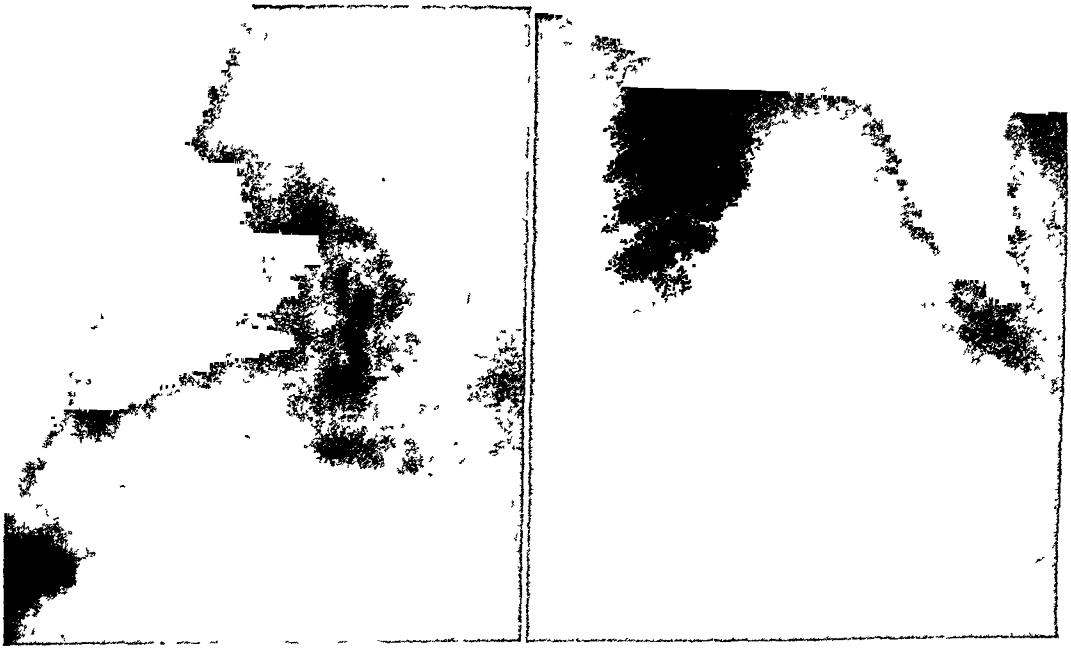


Fig. 1.—Posterior anterior and left lateral view of the chest demonstrating what appears to be a diffuse dilatation of the thoracic aorta

*Laboratory data:* Hemoglobin, 14.8 mg. per cent; hematocrit, 50; sedimentation rate, 16 mm./hr.; white blood count, 17,650; segmented cells, 83 per cent; basophils, 1 per cent; lymphocytes, 9 per cent; and monocytes, 7 per cent. Urinalysis was normal. An electrocardiogram demonstrated a normal sinus rhythm with occasional ventricular extrasystoles. The S-T segments were depressed in all leads, and the T waves were of small amplitude or biphasic in all leads. These studies were interpreted as showing left ventricular strain and myocardial damage. Routine x-ray studies showed a generalized dilatation of the distal aortic arch and the descending aorta.

On Sept. 7, 1955, an aortogram was done, using 70 per cent Diodrast administered through a catheter inserted into the left brachial artery. It showed that there was less than the expected amount of luminal dilatation of the aorta but that a double contrast shadow began at the point of origin of the innominate artery and extended parallel with the left half of the aortic arch and the descending aorta (Fig. 2). This double shadow was on the superior margin of the arch. This demonstration was considered confirmatory of the clinical diagnosis of dissecting aneurysm.

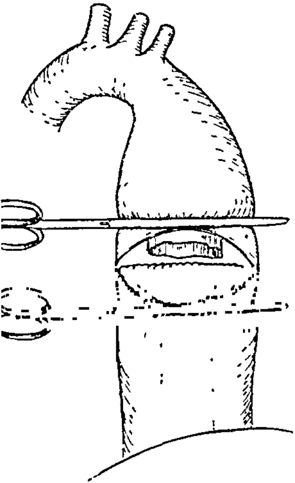


Fig 5

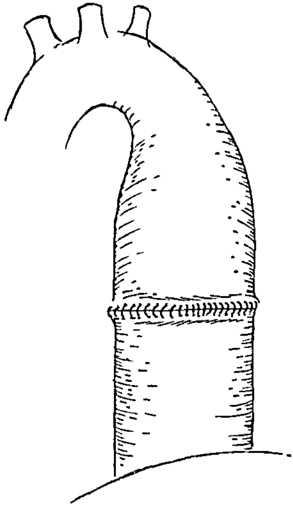


Fig 6

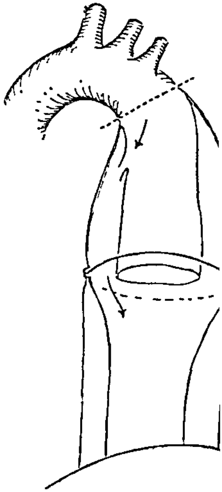


Fig 7

Fig 5—The distal split coats are sutured together with a continuous over and over suture of No. 4-0 arterial silk This effectively reconstitutes the distal aorta

Fig 6.—Proximal outer coats of the dissected aortic wall are now sutured to the entire distal reconstituted wall with No 4-0 arterial silk with a simple over-and-over stitch

Fig. 7—Diagram showing blood flowing proximally in both the outer dissected channel and in its true path into the reconstituted distal aorta

## DISCUSSION

The method of creating a complete "double-barreled" aorta with establishment of a completely free continuity between the true and false lumina at a level well below the site of initial dissection represents a logical modification and extension of the method devised by DeBakey, Cooley, and Creech. By diverting the entire aortic stream into the lumen of the reconstituted lower segment, it would seem that any tendency of a created intimal window to become narrowed, due to clotting or healing, should be eliminated.

The initial attempt to substitute a homologous graft for the dissected aorta in this case led to the suggestion of a possible method of attack upon those relatively numerous dissections which remains limited to the ascending aorta or arch.

By the application of a modified Satinsky clamp of the type used to isolate a portion of the aorta for the attachment of a pericardial pouch for transaortic commissurotomy (Fig. 8), a portion of the outer dissected layer might be excluded and then incised (Fig. 9). Then, a pericardial pouch could be affixed to the lips of the incision. Through this, an exploring finger could be inserted to determine the extent of the dissection accurately (Fig. 10). With a hooked knife, such as was used originally in the performance of mitral commissurotomy, strong suture line. The final result was the creation of two separate tubes proximately emptying into one combined distal tube. The area of the suture line then was reinforced with a circumferentially placed cuff of nylon fabric (Fig. 7). The time of circulatory occlusion was exactly 30 minutes. During the operative procedure, the patient was given *Arfonad* to maintain his arterial pressure at a level of about 70 mm. Hg, systolic.

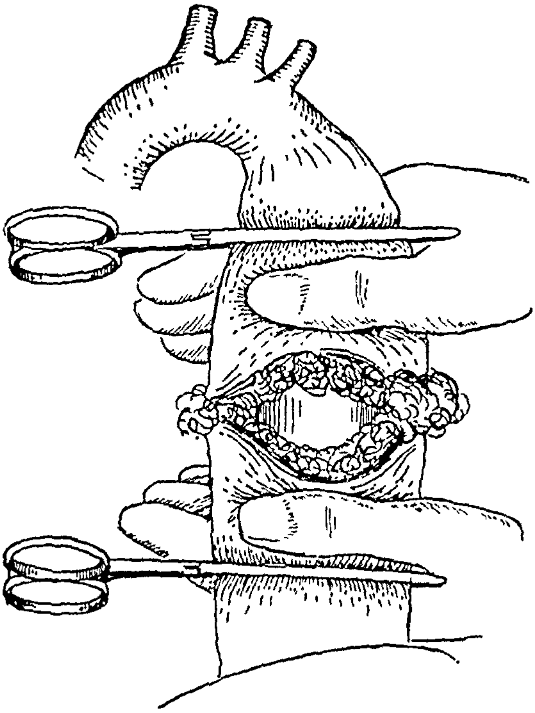


Fig. 3.

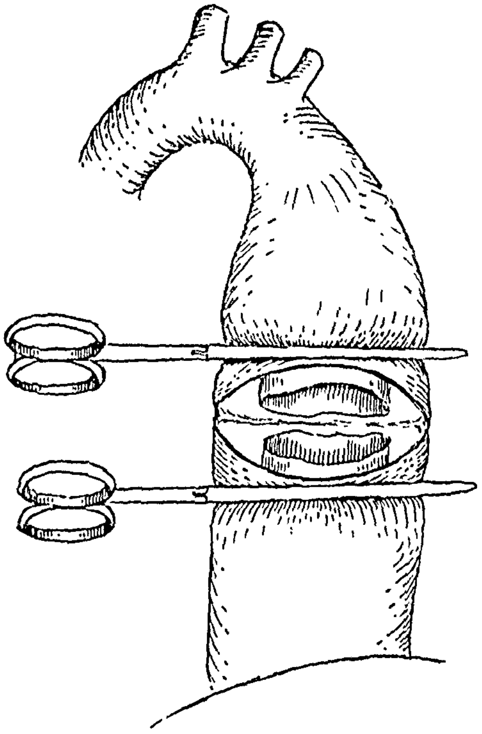
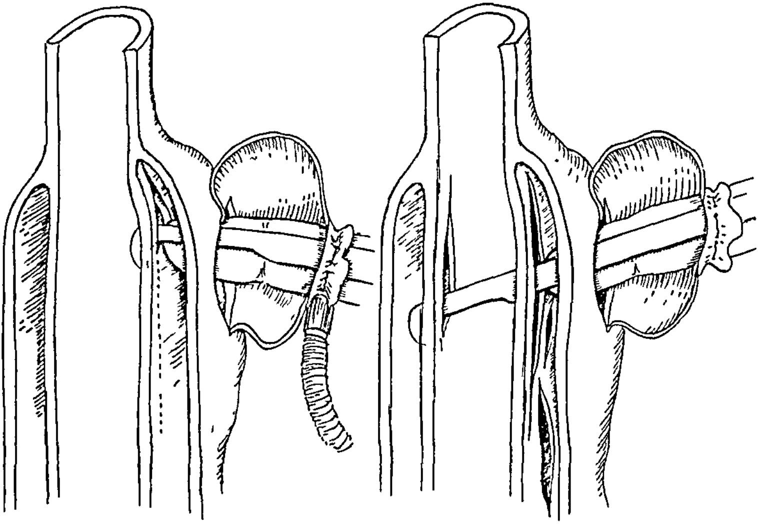


Fig. 4.

Fig. 3.—The outer coats of the dissected wall have been divided transversely leaving the posterior wall of the outer coats intact. The inner tube remains intact at this stage. By gently loosening the clamps and milking the aorta, the clot in the outer channel is removed.

Fig. 4.—The inner tube has been divided transversely throughout its entire circumference.

*Postoperative course:* The patient responded well from the anesthesia. During the first 24 hours, a persistent hypotension was managed with l-norepinephrine and whole blood. In the first 24 hours, there was an output of 240 c.c. of urine, which in the next 48 hours increased to 40 c.c./hr. After the first 24 hours, the patient's circulation became stabilized, and his course was satisfactory. The patient was placed on anticoagulants 12 hours after surgery and these were continued until the twelfth day. Check x-rays the day after surgery showed a small collection of fluid in the left pleural space which was proved to be dark unclotted blood (by thoracentesis). On Sept. 21, 1955, x-ray again revealed evidence of fluid in the pleural cavity. This time 200 c.c. of clear straw-colored fluid was removed. No further fluid accumulated. The patient's progress was slow but he continued to show improvement until Oct. 5, 1955. He then started to vomit and developed anorexia. During the next few days, a progressive increasing jaundice appeared. Cephalin flocculation was 4 plus, thymol turbidity 3.7 units, alkaline phosphatase was 4.3 units, total bilirubin was 12.6 mg. per cent, urobilinogen was present in the urine, and the stools were not acholic. The liver was enlarged and tender. A diagnosis of infectious hepatitis was made and the patient treated for it. His jaundice subsided and his appetite improved. No further difficulties were encountered and the patient was discharged in good condition to his home on Oct. 28, 1955. He has continued to improve, and is quite well at the time of submission of this manuscript for publication.



*A* *B*

Fig 12 —*A*, A hooked knife (original knife used for mitral commissurotomy) is inserted alongside the finger incising the inner tube from the extent of the dissection proximally *B*, Since it is felt that one incision is inadequate to provide free re-entrance of the blood into the true channel multiple splits are made into the true channel

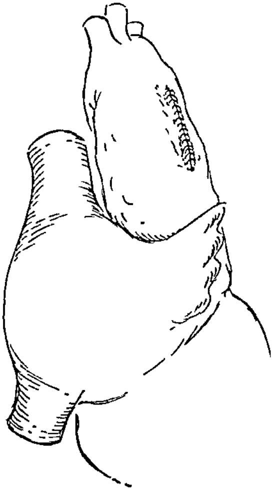


Fig. 13

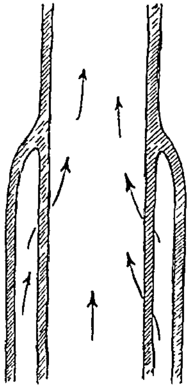


Fig 14

Fig 13 —The clamp has been reapplied the pericardial pouch removed and the incision in the outer coats repaired

Fig 14 —Diagram showing blood coursing in both the true and false channels into the undissected distal channel

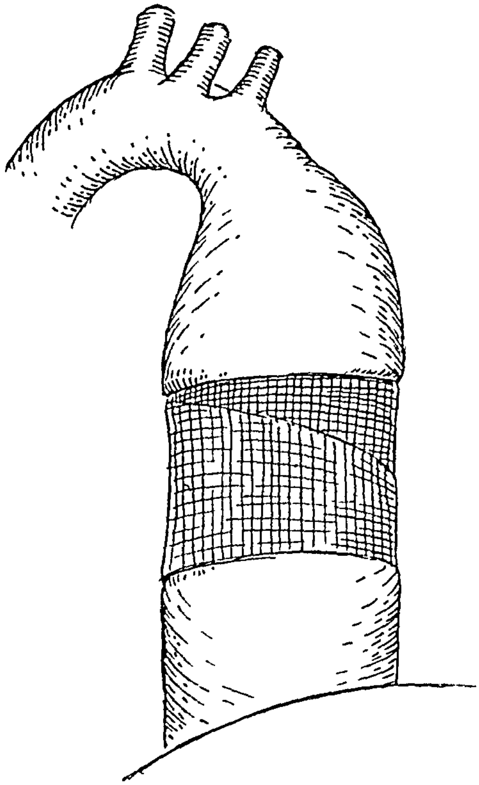


Fig. 8.

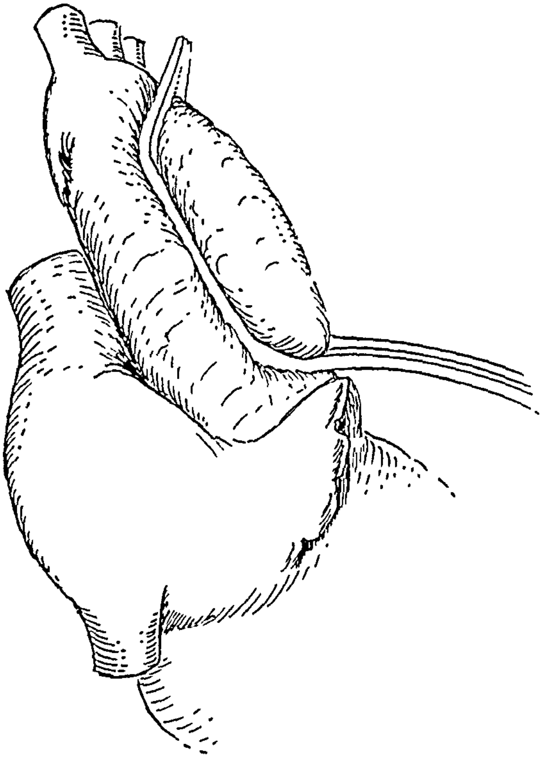


Fig. 9.

Fig. 8.—The entire area of the suture line is wrapped with nylon for reinforcement.

Fig. 9.—A portion of the dissected ascending aortic wall excluded by a modified Satinsky clamp.

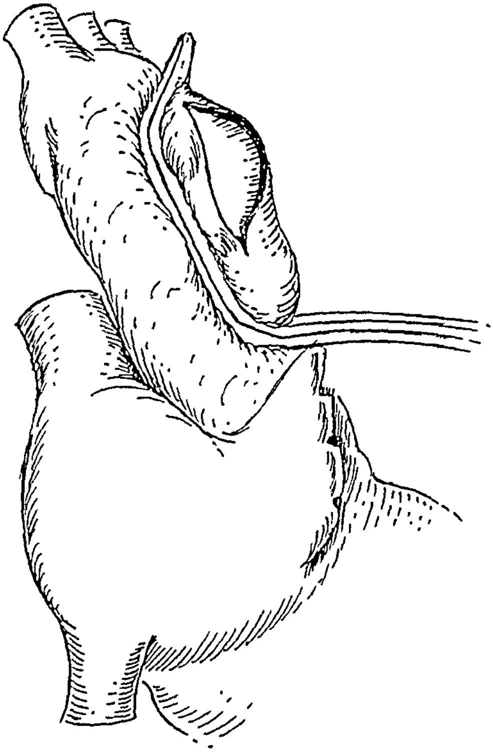


Fig. 10.

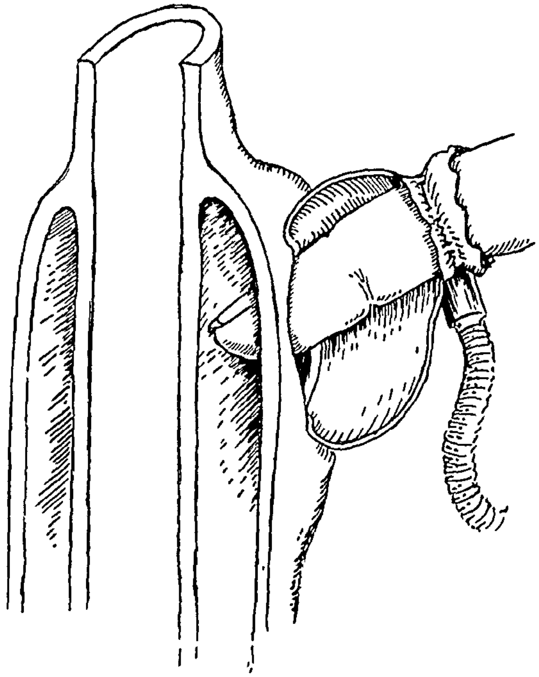


Fig. 11.

Fig. 10.—The outer coats have been incised but the inner tube remains intact.

Fig. 11.—A pericardial pouch has been attached to the outer dissected wall (after the manner of attaching a pericardial pouch for aortic commissurotomy) and a finger is inserted to explore the extent of the dissection.

# FAILURE OF CARDIO-PERICARDIOPEXY TO PROTECT PIGS AGAINST ACUTE CORONARY OCCLUSION

Harry Gross, M.D., Allan E. Bloomberg, M.D , Morton Rosenblatt, M.D.,
Adrian Kantrowitz, M.D., Clarence J. Schein, M.D., and
Ruthven Ferreira
New York, N. Y.

THE purpose of this study was to determine whether the operation of talc cardio-pericardiopexy would protect the pig against acute coronary occlusion. This animal was chosen for two reasons. First, Blumgart and his coworkers showed that the pig heart, like the human, normally has no intramyocardial anastomoses.[1] Second, the rapid growth of the pig in a relatively short period of time makes it possible to evaluate the protective role of talc during a period of rapid growth. Chardack's[2] recent work in the dog further emphasizes the fallacy of using dogs in the study of experimental coronary disease.

## METHOD

Yorkshire piglets weighing from 30 to 60 pounds were anesthetized with either intravenous or intraperitoneal veterinary Nembutal. An endotracheal airway was established in all animals. Our earlier attempts were directed at transglottic intubation using specially devised long laryngoscopic blades and tubes. This procedure was not uniformly successful. The later experiments were carried out via transtracheal intubation. In all animals requiring intubation, positive pressure oxygen was administered.

*Control Group.*—Following intubation, the heart was exposed transpleurally in a group of 6 piglets. The left anterior descending coronary artery was tied 2 cm. or more from its origin. Many of these animals survived. For this reason, the left anterior descending coronary must be ligated within 2 cm. of its origin in order uniformly to kill the animal.

*Experimental Group.*—Animals were prepared by inserting 4 to 6 Gm. of sterile USP talc (magnesium silicate) into the pericardium. This was done as an extrapleural procedure. All animals received antibiotics postoperatively.

Fifty pigs were used in this phase of the experiment. Twenty-one survived the procedure and were challenged at various intervals from one to fifty-five weeks. The remaining animals were included in the control group used to determine the site of lethal ligation, or were lost due to technical difficulties. The challenge entailed left anterior coronary artery ligation at the site which

---

From the Medical and Surgical Divisions of the Montefiore Hospital, New York City, N. Y.
Received for publication May 7, 1956.

inserted alongside the finger into the pouch, the inner layer then might be repeatedly incised longitudinally to the most distal limit of the dissection (Figs. 11, 12 *A* and *B*, and 13). By reapplication of the clamp, the pouch would be removed and the outer layer of the wall repaired with sutures. This area then might be reinforced with a layer of nylon fabric. Thus, conversion to a "healed aneurysm" would be brought about (Fig. 14).

This would not reconstitute the aorta distal to the new opening, but would tend to alleviate the effective head of dissecting pressure by allowing a run-off into the natural aortic lumen. It is brought to mind that these dissections in this area apparently reach a zone in the media which is resistant to the dissecting force so that frequently they do not progress further but rupture either outward or inward before progression can be resumed. Unfortunately, most often they rupture to the exterior. By the creation of a large vent through which the blood may return to its normal channel, at least the effective head of the dissecting column will be reduced. This will not be complete assurance against further progression, but would seem to be at least a step forward in the treatment of this very large group of dissections which do not progress far enough for more completely definitive surgery before external rupture occurs.

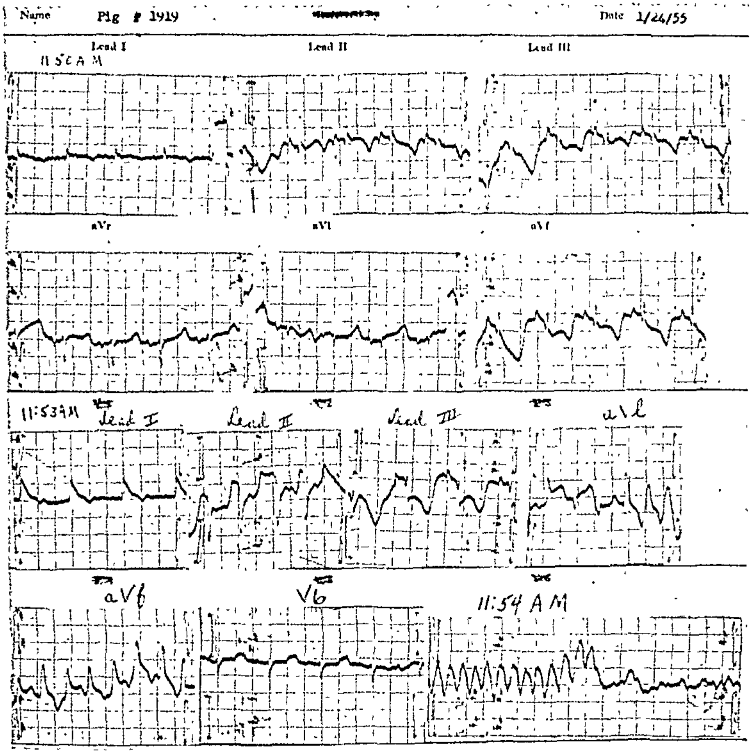
## SUMMARY AND CONCLUSIONS

1. The surgical concept of creating a "healed aneurysm" of the aorta has been discussed.
2. A case with survival after the fullest exhibition of such surgical treatment has been presented.
3. A modification of the present method of treatment was used which consisted of leading the two entirely separate lumens into a reconstituted distal segment, thus accomplishing entire diversion of the dissection pressure head.
4. A possible method of corrective approach to dissections which occur only within the arch and ascending aorta is presented.

## REFERENCES

1. DeBakey, M. E., Cooley, D. A., and Creech, O.: Aneurysms and Occlusive Diseases of the Aorta: Analysis of 203 Cases Treated by Resection and Homograft Replacement, Henry Ford Hospital International Symposium on Cardiovascular Surgery, Philadelphia, 1955, W. B. Saunders Company, p. 468-495.
2. DeBakey, M. E., Cooley, D. A., and Creech, O.: Surgical Considerations of Dissecting Aneurysm of the Aorta, Ann. Surg. 142: 586-612, 1955.
3. Erdheim, J.: Medionecrosis Aortae Idiopathica, Virchows Arch. f. path. Anat. 273: 454-479, 1929.
4. Erdheim, J.: Medionecrosis Aortae Idiopathica Cystica, Virchows Arch. f. path. Anat. 276: 187-299, 1930.
5. Gurin, D., Bulmer, J. W., and Derby, R.: Dissecting Aneurysm of the Aorta, New York State J. Med. 35: 1200-1202, 1935.
6. Levinson, D. C., Edmeades, D. T., and Griffith, G. C.: Dissecting Aneurysm of the Aorta: Its Clinical Electrocardiographic and Laboratory Features, Circulation 1: 360-387, 1950.
7. Moritz, A. R.: Medionecrosis Aortae Idiopathica Cystica, Am. J. Path. 8: 717-734, 1932.
8. Shaw, R. S.: Acute Dissecting Aortic Aneurysm: Treatment by Fenestration of the Internal Wall of the Aneurysm, New England J. Med. 253: 331-333, 1955.
9. Shennan, T.: Dissecting Aneurysms, Medical Research Council, Great Britain National Health Insurance Medical Research Committee, Special Report Series 190-199, p. 7-138, His Majesty's Stationery Office, London, 1934.
10. Weiss, S., Kinney, T. D., and Maher, M. M.: Dissecting Aneurysm of the Aorta With Experimental Atherosclerosis, Am. J. M. Sc. 200: 192-203, 1940.

*Microscopic Findings.*—Sections were taken from the mid- and posterior portions of the interventricular septum and the left anterior ventricular wall. Histologic examination of the specimens showed two types of reaction. In the early cases there was acute pericarditis with an inflammatory reaction. In the later cases, there were scarring and foreign-body granuloma formation with numerous giant cells. In none was there a significant or widespread collateral circulation into the pericardium or myocardium (Figs. 1-4).



Pig # 1919

Ligation of left main coronary artery is followed by rapid changes terminating in ventricular fibrillation four minutes later.

Lead I shows inversion of T waves.
Lead II shows sharply pointed deeply inverted T waves.
Lead III shows RT depression and sharply pointed deeply inverted T waves.
Lead aVf shows profound RT depression.
Three minutes after ligation:
Lead I shows a broadening of the R wave.
Lead II shows a marked shift in axis and marked widening of QRS.
Lead III shows marked RT depression and inverted T waves.
Lead aVf shows multifocal premature beats followed by a run of ventricular p beats.
Four minutes after ligation fatal ventricular fibrillation developed.

had proved fatal in the control group. These operations were done transpleurally. During the survival period which ranged from one to fifty-five weeks, the older animals increased their weight tenfold so that the largest survivors weighed 600 to 700 pounds.

The anterior left coronary artery was ligated and divided within 2 cm. of its origin. In a few animals, intermittent occlusion by a clamp preceded the final ligation and division of the artery. Continuous electrocardiogram tracings were obtained on all the animals.

## EXPERIMENTAL RESULTS

The results are detailed below. It is important to note that in the interval between talc preparation and challenge, there was no interference with the growth or activity of the animal. In no instance was constrictive pericarditis observed.

The electrocardiographic changes resulting from talc were characterized by transitory evidence of pericarditis and myocarditis in the form of lowering of the T waves. The electrocardiographic changes described were all evaluated in relation to the preoperative record. Electrocardiograms taken immediately following ligation of the coronary arteries showed the typical responses to coronary ligation with the development of currents of injury, displacement of the RT or QT segments up or down, the development of T-wave changes, and various types of arrhythmias. Monophasic curves of injury were frequent. Premature beats of the auricle or ventricle, often multifocal, developed; runs of premature ventricular beats that were often the precursors of fatal ventricular tachycardia. In many instances, the transition from normal sinus rhythm to fatal ventricular tachycardia was direct and sudden. In the cases where the occlusion was temporary and released before the onset of ventricular fibrillation, it was possible to restore normal sinus rhythm and to abolish currents of injury.

The period of survival following ligation in the talc-prepared animals paralleled exactly that of the control animals. No animal survived the procedure. The maximum period of survival was 29 minutes.

*Pathologic Findings.*—The ligation of the left anterior descending coronary artery within 2 cm. of its origin was confirmed in all cases. Since the time between occlusion of the coronary vessel and the death of the animals was short, none of the hearts exhibited gross changes in the myocardium. All showed a grayish-white thickening of the pericardium. In the hearts of pigs surviving one to four weeks after talc installation, there was a fibrinous exudate on the surface of the thickened pericardium. The hearts of longer survivals showed more thickening. The pericardial reaction was greater over the anterior than over the posterior part of the ventricles. Over the posterior surfaces of some specimens, fairly normal pericardium was found. The pericardial reaction was greater near the atrioventricular groove than at the apex. Both of these differences are related to the anterior injection of the talc.

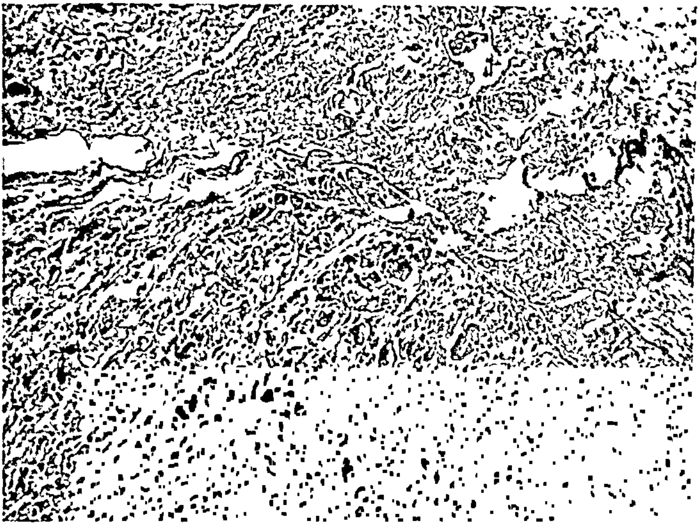


Fig. 3.—A section through the pericardium over the left anterior ventricular wall in a pig that survived five months before the coronary artery was ligated Note the dense fibrosis surrounding the talc granules, an occasional foreign-body cell, and a very few small blood vessels.

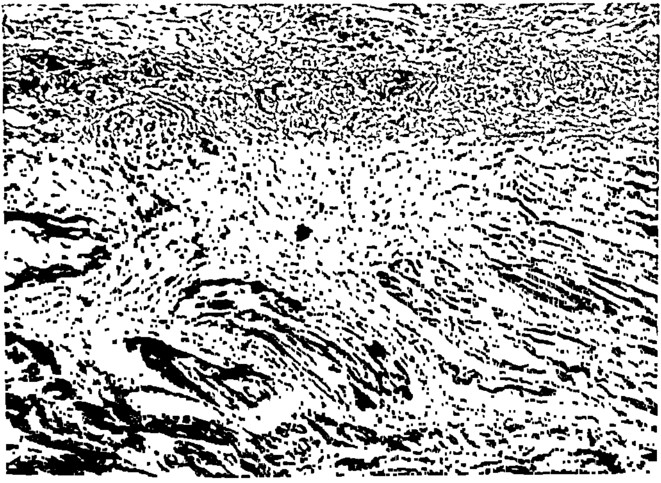


Fig. 4.—This section shows both the pericardium and myocardium from the left anterior ventricular wall in another pig that survived five months. Again note the dense fibrosis in the pericardium with the lack of new blood vessel formation in either the pericardium or myocardium

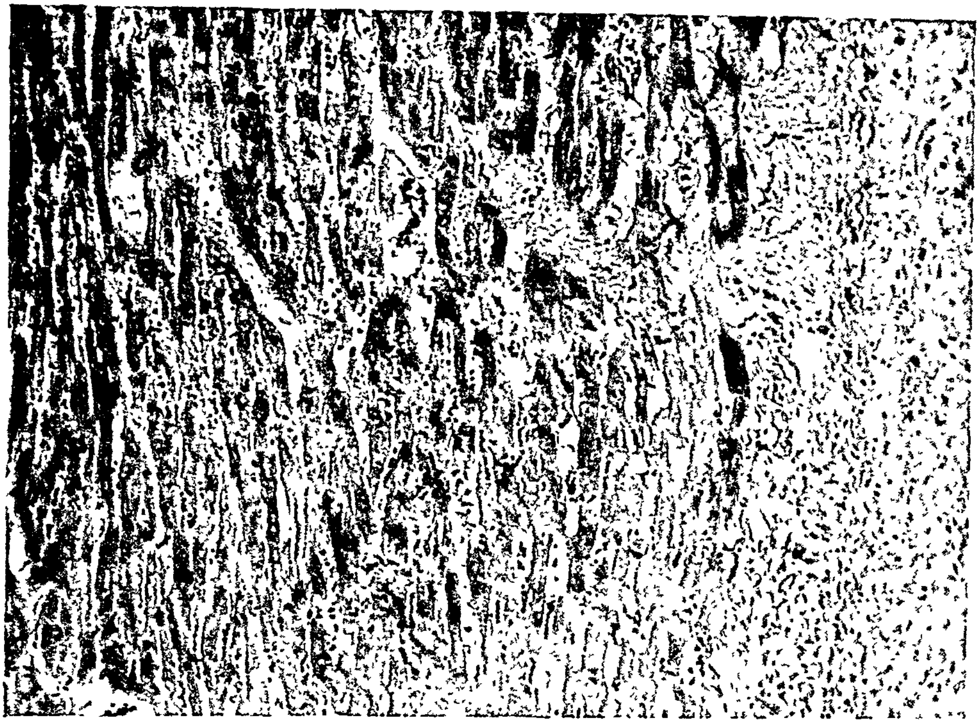


Fig. 1.—Section through pericardium and myocardium (left anterior ventricular wall) of a one-week specimen. *Note thickening in pericardium and lack of new small blood vessel formation in either the pericardium or myocardium.*

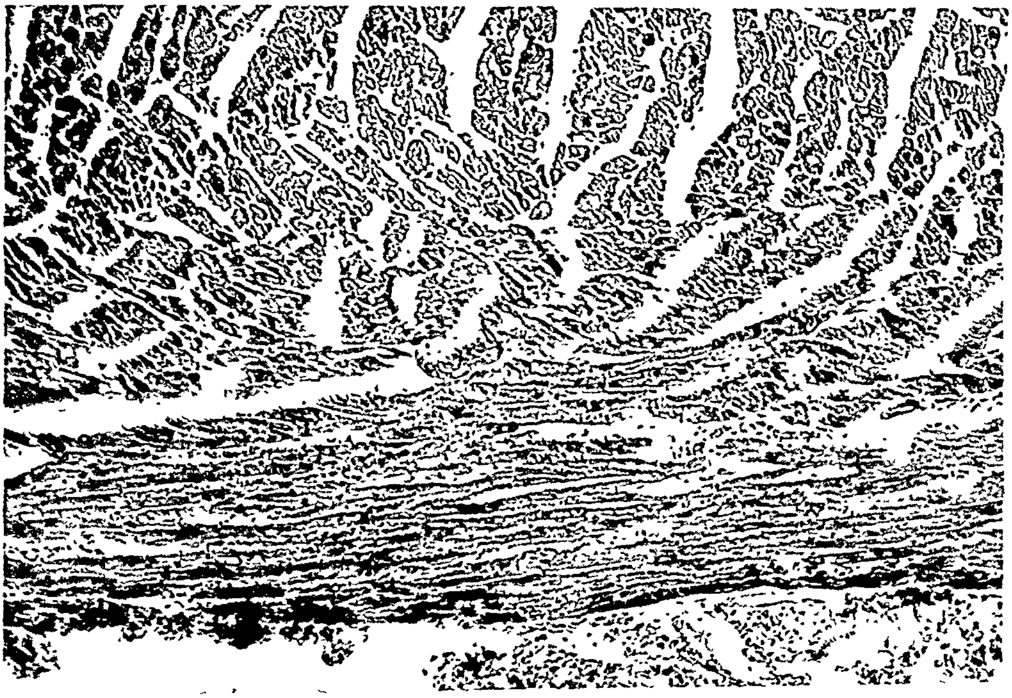


Fig. 2.—Similar section in another one-week specimen. Again note lack of blood vessels in myocardium directly beneath pericardium involved in the talc reaction.

# AN EVALUATION OF TWO NEW TOPICAL ANESTHETIC AGENTS: DYCLONE* AND CYCLAINE**

F. A. Hughes, M.D., J. D. Mobley, M.D., R. J. Bills, M.D.,
J. R. Burwell, M.D., and J. W. Pate, M.D.
Memphis, Tenn.

THE "ideal" topical anesthetic agent should possess the following properties (1) rapid onset of anesthesia (2) high potency, (3) freedom from untoward reactions in amounts normally employed, (4) wide margin of safety even when absorbed, (5) no local irriation, and (6) no cross sensitization to other anesthetic agents. This paper presents a comparison of two new agents developed in the continuing search for the ideal topical anesthetic agent.

The present study is divided into two parts. The first is a review of the literature on the toxicity of Dyclone and Cyclaine and our toxicity studies on Dyclone, Cyclaine, tetracaine, and cocaine. The second part consists of a clinical evaluation of Dyclone and Cyclaine anesthesia for bronchoscopy, bronchography, esophagoscopy, and esophageal dilatation.

## REVIEW OF THE LITERATURE

Beyer, Latven, Freyburger, and Parker[1] compared the toxicity of Cyclaine, cocaine, and other anesthetic agents by the $LD_{50}$ technique. The findings of their study of the toxicity of Cyclaine and cocaine in milligrams per kilogram of body weight are shown in Table I. It is apparent that these two agents are of equal toxicity when given intravenously in mice but, when administered subcutaneously Cyclaine is considerably less toxic, milligram for milligram, in mice, rabbits, and guinea pigs. No report of Cyclaine toxicity studies in dogs could be found. Beyer and co-workers[1] found that as a topical anesthetic agent, Cyclaine is as potent as cocaine when equal concentrations are employed. They also state that 1 and 2 per cent concentrations of Cyclaine give approximately the same duration of anesthesia as the corresponding concentrations of cocaine.

TABLE I

| | MICE | | RABBITS | GUINEA PIGS | |
|---|---|---|---|---|---|
| AGENT | IV (MG./KG.) | SUBCUTANEOUS (MG./KG.) | SUBCUTANEOUS (MG./KG.) | AQUEOUS SOLUTION (MG./KG.) | ADRENALIN SOLUTION (MG./KG.) |
| Cyclaine | 24 | 1080 | 164 | 146 | 385 |
| Cocaine | 25 | 140 | 70 | 27 | 87 |

---

From the Thoracic Surgery Section Veterans Administration Medical Teaching Group Hospital, Memphis 15, Tenn.

Received for publication May 8, 1956.

*Dyclonine hydrochloride—Pitman-Moore Company, Indianapolis 6, Ind.

**Hexylcaine hydrochloride—Sharp & Dohme Division of Merck & Co., Inc. Philadelphia 1, Pa.

## DISCUSSION

No beneficial effects resulted from talc cardio-pericardiopexy in protecting the pig heart against subsequent ligation of the coronary arteries.

Pericardial adhesions developed wherever talc powder was insufflated. The adhesions were dense and so avascular that when they were cut, no bleeding resulted. They were firm enough so that they had to be cut by sharp dissection for, when cut by blunt dissection, atrial tears frequently resulted.

During the period of observation, there was no histologic or gross evidence of increased collateral circulation. The experimental animals did not survive any longer than the controls regardless of the duration of talc instillation.

The burden imposed on the coronary circulation by our experiments was great. The ligations were sudden, complete and high, 2 cm. or less distal to the bifurcation of the left coronary artery. Since there was no stimulus for the development of a coronary collateral circulation in our animals by progressive coronary narrowing as in Blumgart's experiments, the burden on the coronary circulation and the test of the effectiveness of pericardio-pericardiopexy were great.

The experimental conditions do not completely mimic the course of human coronary artery disease. In man, the greatest stimulus to a collateral circulation is progressive coronary narrowing. Indeed, when the collaterals are well developed, occlusions of one or both major coronary arteries may be well tolerated. Contrariwise, sudden hemorrhage into a plaque of a coronary artery close to the bifurcation, when coronary disease is local, is usually fatal in man. This clinical condition is simulated in the experiments performed.

## SUMMARY

1. In the normal pig, the introduction of talc into the pericardium is well tolerated.

2. Animals surviving talc pericardio-cardiopexy for as long as one year were not protected by this operation against acute occlusion of the left coronary artery.

3. Constrictive pericarditis was not produced in any of the animals.

We are indebted to the Lederle Laboratories Division of the American Cyanamid Company without whose generous help this work could not have been carried out, and to Drs. Kanegis, Percival, and Tonelli of their Staff for much help.

## REFERENCES

1. Blumgart, H. L., Zoll, P. M., Freedberg, A. S., and Gilligan, D. R.: The Experimental Production of Intercoronary Arterial Anastomoses and Their Functional Significance, Circulation 1: 10, 1950.
2. Chardack, W. M., Bolgan, F. J., Olson, K. C., Gage, A. A., and Farnsworth, W. E.: The Mortality Following Ligation of the Anterior Descending Branch of the Left Coronary Artery in Dogs, Ann. Surg. 141: 443, 1955.

just listed at the rate of 1 c c per minute until dead  The end point of death was determined as the time of cessation of the heartbeat  The lethal dose for the various agents in milligrams per kilogram of body weight is shown in Table II

TABLE II

| AGENT | AVERAGE LETHAL DOSE | | |
|---|---|---|---|
| | UNANESTHETIZED (MG /KG ) | ANESTHETIZED (MG /KG ) | COMBINED GROUPS (MG /KG ) |
| Cyclaine (6 dogs) | 32 0<br>(3 dogs) | 31 9<br>(3 dogs) | 31 95 |
| Dyclone (6 dogs) | 12 9<br>(3 dogs) | 11 8<br>(3 dogs) | 12 3 |
| Tetracaine (4 dogs) | 11 0<br>(2 dogs) | 6 2<br>(2 dogs) | 8 4 |
| Cocaine (4 dogs) | 22 4<br>(2 dogs) | 28 4<br>(2 dogs) | 25 0 |

The picture appears different (Table III) when these same data are considered in volume of each agent per kilogram of body weight  Thus, to obtain a lethal dose in dogs it required 3 8 times as great a volume of Dyclone per kilogram as Cyclaine, 5 9 times the volume of Dyclone as tetracaine, and 10 times the volume of Dyclone as cocaine  Cyclaine appears slightly less toxic (c c /Kg ) than tetracaine and cocaine, but the difference here is not great, and the number of dogs employed was not large enough to be statistically significant  The comparison on the basis of cubic centimeters per kilogram is more valid because the various agents were used in the concentrations that are used clinically

TABLE III

| AGENT | AVERAGE LETHAL DOSE | | |
|---|---|---|---|
| | UNANESTHETIZED (C C /KG ) | ANESTHETIZED (C C /KG ) | COMBINED GROUPS (C C /KG ) |
| Cyclaine | 0 64 | 0 65 | 0 65 |
| Dyclone | 2 6 | 2 4 | 2 5 |
| Tetracaine | 0 55 | 0 31 | 0 42 |
| Cocaine | 0 22 | 0 28 | 0 25 |

All of the unanesthetized dogs had strikingly similar responses following administration of these various anesthetics  All dogs exhibited marked salivation, urinary incontinence, generalized convulsions, and usually hyperpnea prior to cessation of respiration  In nearly all cases, respiration ceased a few seconds prior to the cessation of heartbeat

In the anesthetized animals pulse and arterial pressure were recorded by means of a catheter in the femoral artery connected to a mercury manometer and kymograph  Each cubic centimeter dose of Cyclaine, tetracaine, and cocaine produced in all animals an almost immediate marked fall in pressure that persisted for approximately 30 seconds following administration  Following Dyclone administration the pressure and pulse remained nearly constant until the lethal dose was approached at which time the pressure fell

Hudson and Jacques[2] report the use of Cyclaine topically for a variety of procedures, including bronchoscopy, with a negligible incidence of untoward reactions. Clark, Orkin, and Rovenstine[3] state that a 2.5 per cent solution of Cyclaine will anesthetize the larynx in almost every patient for a period of 15 minutes and a 5 per cent solution is effective in all instances for approximately 30 minutes.

Abreu and his associates[4] report that 2 mg./Kg. body weight of Dyclone administered intravenously in anesthetized dogs did not significantly affect blood pressure or pulse, nor did it reduce the cardiovascular response to acetylcholine or increase response to epinephrine, demonstrating a lack of parasympatholytic action. Doses of 5 to 10 mg./Kg. in anesthetized dogs given in a period of one to 2 minutes caused respiratory failure in some animals. In 15 unanesthetized dogs, they found that the dogs tolerated 2.5 to 3.0 mg./Kg. intravenously and 20.0 to 25.0 mg./Kg., orally. The intravenous $LD_{50}$ for dogs was found to be 9.5 ± 1.3 mg./Kg.

Arora and Sharma[5] found 10 mg./Kg. Dyclone fatal to anesthetized dogs when administered in a period of 25 seconds. These investigators describe the pharmacology of Dyclone in detail. Arora, Kulshrestha, and Sharma[6] found Dyclone in a 0.5 per cent concentration to be more rapid in onset and of about equal potency as 5 per cent cocaine or 1 per cent tetracaine when used topically for ophthalmic surgery.

Himalstein,[7] in a recent review of the problem of topical anesthesia prior to peroral endoscopy, concluded that severe and fatal reactions are far more common than generally appreciated. His opinion was that most fatalities are due to overdosage and only a small number due to true drug idiosyncrasy. He points out the wide range of dosage of cocaine and tetracaine employed by different endoscopists. He stressed the importance of using as small a quantity of anesthetic as possible commensurate with adequate anesthesia.

In his experience, lidocaine and Cyclaine showed some promise of supplanting cocaine.

## METHOD OF STUDY AND RESULTS

We undertook to determine the intravenous lethal dose of both Cyclaine and Dyclone in dogs and to compare the lethal dose of these agents with those of cocaine and tetracaine similarly administered. We employed the various anesthetic agents at the concentrations used clinically preparatory for endoscopy. Previously at the Veterans Administration Medical Teaching Group Hospital (Kennedy), Memphis, Tenn., we have employed either 10 per cent cocaine or 2 per cent tetracaine for anesthesia prior to endoscopy. Presently, we are employing 5 per cent Cyclaine or 0.5 per cent Dyclone.

In the experimental laboratory, 20 healthy mongrel dogs weighing between 8.2 and 20 kilograms were used, one-half of which were unanesthetized and one-half were lightly anesthetized with intravenous sodium pentobarbital. All received the selected anesthetic agent intravenously in the concentrations

## SUMMARY

From the data presented, it is apparent that, in dogs, intravenous Dyclone is appreciably less toxic than Cyclaine, tetracaine, or cocaine. Apparently Cyclaine in dogs is slightly less toxic than tetracaine and cocaine, but the difference is not great. Clinically, mild troublesome reactions are less frequent with these agents than they were when tetracaine and cocaine were employed.

Both Cyclaine and Dyclone produce adequate anesthesia in most patients for about 30 to 40 minutes. Further clinical use of these agents is warranted.

## REFERENCES

1. Beyer, K. H., Latven, A. R., Freyburger, W. A., and Parker, M. P.: A Comparative Study of the Activity and Toxicity of Hexylcaine (1-Cyclohexylamino-2-Propyl benzoate), A New Local Anesthetic Agent, J. Pharmacol. & Exper. Therap. 93: 388-400, 1948.
2. Hudson, F., and Jacques, A.: Proc. Canad. Anesthetists Soc., 1952, p. 34.
3. Clark, R. E., Orkin, L. R., and Rovenstine, E. A.: To be published.
4. Abreu, B. E., Richards, A. B., Weaver, L. C., Burch, G. R., Bunde, C. A., Bockstahler, E. R., and Wright, D. L.: Pharmacologic Properties of 4-Alkoxy-β-(1-Piperidyl) Propiophenones, J. Pharmacol. & Exper. Therap. 115: 419-426, 1955.
5. Arora, R. B., and Sharma, V. N.: Local Anesthetic and Pharmacologic Properties of 4-n-Butoxy-β-(1-Piperidyl) Propiophenone HCl and β-Diethylaminoethyl-p-n-Hexyloxybenzilate HCl, J. Pharmacol. & Exper. Therap. 115: 413-418, 1955.
6. Arora, R. B., Sharma, V. N., and Kulshrestha, P. P.: Clinical Trial of Dyclonine in Intraocular Ophthalmic Surgery, Eye, Ear, Nose & Throat Month. 34: 593, 1955.
7. Himalstein, M. R.: The Problem in Endoscopic Topical Anesthesia, A. M. A. Arch. Otolaryng. 63: 60-66, 1956.
8. Hughes, F. A., Burwell, J. R., and Pate, J. W.: The Use of a New Topical Anesthetic Agent (Dyclone) in Peroral Endoscopy: A Report of 569 Procedures, J. Thoracic Surg. 32: 135, 1956.

Salivation, urination, and convulsion were totally absent in the anesthetized animals. These animals did, however, exhibit hyperpnea just prior to cessation of respiration as did the unanesthetized dogs.

## CLINICAL EVALUATION

Previously, patients who were to undergo endoscopy, bronchography, or dilatation of esophageal strictures were anesthetized with approximately 10 c.c. of a 10 per cent cocaine solution or a 2 per cent tetracaine solution. Approximately 7 c.c. of either agent was used to anesthetize the throat and larynx by applying the agent to the pyriform fossa with Jackson cross-action forceps. Approximately 3 c.c. of the remaining anesthetic was diluted with water to form solutions of 1 per cent and ½ per cent, respectively. The diluted anesthetic was then dripped between the vocal cords in aliquots of 1 to 2 c.c. until adequate anesthesia of the bronchial mucosa was obtained. Patients who were to undergo bronchoscopy or bronchography were instructed not to swallow any of the agent so as to minimize unnecessary absorption. If the patient was to undergo esophagoscopy or esophageal dilatation, small quantities of the diluted anesthetic agent were swallowed. Using this technique, adequate anesthesia was consistently obtained in over 6,000 successive endoscopies during the years 1943 to 1954, with no fatalities attributable to topical anesthesia. Serious untoward reactions were few; however, mild, disconcerting anesthetic reactions occurred in approximately 1 or 2 per cent of cases.

As a clinical research project, we started employing Dyclone for anesthesia in July, 1954, and Cyclaine in April, 1955. Fifteen cubic centimeters or less of either 5 per cent Cyclaine or 0.5 per cent Cyclone is employed by the same technique as described for the other agent. Since April, 1955, the nurse has set up the anesthetic trays and has alternated the anesthetic agents so that the endoscopist does not know which anesthetic he is employing until the examination is over. Since Cyclaine and Dyclone have been used we have performed 457 bronchoscopies, 67 esophagoscopies, 289 bronchograms, and 56 esophageal dilatations on 665 patients. Some of these patients were included in a previous report from this hospital on Dyclone topical anesthesia.[8]

Results have been satisfactory with both agents with respect to potency, promptness of action, and relative freedom from untoward reactions. The oxygen tank and the Pentothal syringe now go almost unobserved in the corner of the anesthetic room. After the completion of an examination, the endoscopist is permitted to learn what anesthetic he has employed on his preceding patient. By this method, the impression has been gained that Cyclaine becomes effective a little more promptly than Dyclone. However, the latter produces adequate anesthesia in about six minutes. Some examiners feel that Cyclaine gives more complete anesthesia. The duration of anesthesia with each agent is usually about 30 to 40 minutes which is adequate for most endoscopists. In indicated cases, the anesthesia is usually adequate for subsequent bronchography.

TABLE I

| INITIAL | AGE | SEX | $O_2$ REMOVAL REST (ML./L.) | $O_2$ REMOVAL EXERCISE (ML./L.) | $O_2$ CONSUMPTION REST (ML./MIN.) | $O_2$ CONSUMPTION EXERCISE (ML./MIN.) | VENTILATION REST (LITERS) | VENTILATION EXERCISE (LITERS) | DIAGNOSIS | V.C. (MLS.) | M.B.C. (LITERS) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| L L | 30 | F | 34 | 31 | 268 | 495 | 9.5 | 15.7 | Bronchiectasis of RLL | 3057 | 78 |
| T H | 26 | F | 44 | 33 | 294 | 460 | 6.7 | 13.1 | Tuberculosis of right lung | 1449 | 47 |
| V L | 24 | F | 41 | 16 | 220 | 122 | 5.3 | 26.3 | Pulmonary artery severance | 1694 | 51 |
| R K | 30 | F | 37 | 22 | 276 | 369 | 7.4 | 16.6 | Pulmonary embolus | 2569 | 121 |
| M W | 63 | F | 31 | 19 | 278 | 390 | 9.2 | 20.1 | Bronchogenic carcinoma | 1910 | 70 |
| L H | 61 | M | 38 | 15 | 273 | 291 | 7.1 | 18.7 | Carcinoma of left lung | 2010 | 32 |
| W F | 70 | M | 53 | 80 | 409 | 1050 | 7.8 | 13.1 | Carcinoma of pancreas secondary to lung | 1334 | 38 |
| B A | 59 | M | 26 | 45 | 204 | 338 | 7.8 | 12.0 | Benign teratoma | 4030 | 80 |
| R J | 58 | M | 42 | 40 | 310 | 710 | 7.4 | 17.7 | Carcinoma of LUL (Pancoast) | 3850 | 60 |
| B A | 64 | M | 29 | 29 | 220 | 535 | 7.5 | 19.5 | Renal carcinoma, secondary in lung | | |
| R E | 16 | F | 33 | 33 | 170 | 450 | 5.1 | 12.5 | Benign ganglioneuroma | 3240 | 102 |
| R B | 43 | M | 22 | 15 | 208 | 264 | 9.5 | 15.0 | Mediastinal torsion | | |
| T D | 44 | M | 28 | 19 | 224 | 392 | 8.0 | 21.0 | Tuberculosis of RUL, mediastinal torsion | 2660 | 55 |
| G R | 51 | M | 19 | 38 | 200 | 527 | 10.7 | 13.6 | Tuberculosis of LUL | 3400 | 76 |
| W S | 43 | M | 25 | 27 | 224 | 430 | 9.0 | 16.0 | Mitral stenosis | | |
| D A | 50 | M | 28 | 13 | 203 | 250 | 7.3 | 19.0 | Mitral stenosis | | |
| R G | 46 | M | 26 | 27 | 224 | 317 | 8.3 | 11.6 | Mitral stenosis | | 36 |
| M M | 50 | F | 22 | 17 | 201 | 238 | 9.0 | 13.9 | Mitral stenosis and tricuspid insufficiency | 1912 | 32 |
| K S | 51 | M | 33 | 54 | 213 | 510 | 7.0 | 9.5 | Mitral stenosis and mitral insufficiency | 2800 | |
| M A | 46 | M | 17 | 38 | 208 | 518 | 12.2 | 13.6 | Aortic stenosis | 3174 | 99 |
| D T | 46 | F | 22 | 23 | 222 | 296 | 10.9 | 13.0 | Mitral stenosis and mitral insuf | 2056 | 57 |

# CLINICAL SIGNIFICANCE OF OXYGEN REMOVAL

JOHN C. KOVACH, M.D.,* AND GEORGE MORALES, M.D.**
NEW YORK, N. Y.

ON our surgical cardiac patients, the routine preoperative work-up includes cardiac catheterization, residual lung capacity determination, angiocardiography, and spirometric studies. In the course of investigation, it has become evident that the oxygen removal is of special significance. A low value with no increase or a decrease on exercise may have ominous portent.

Two postoperative fatalities in young patients after pulmonary resection (lobectomy and pneumonectomy) who had had no apparent abnormalities beyond their pulmonary disease preoperatively, other than an abnormal oxygen removal, focused our attention on this entity.

The oxygen removal is the amount of oxygen in milliliters that is removed from one liter of respired air. The normal removal rate at rest is 40 ml. per liter of ventilation and at exercise is 60 ml., an increase of 50 per cent. Normal resting and exercise ventilation rates are assumed to be 5 and 15 L. per minute for an average man.

## CLINICAL MATERIAL

To illustrate the various causes of a low oxygen removal, we have selected the following case histories. Table I summarizes the data in the order discussed.

The only abnormal feature of the first patient (L. L.) was a low oxygen removal, 34 ml. per liter of ventilation, which did not increase on exercise. Clinically, with all available evidence, she was considered to have a normal heart, there was no hyperventilation and there was a straightforward diagnosis of right lower lobe bronchiectasis without sequelae. The chest plate was essentially normal (no parenchymal disease). Preoperatively, the low oxygen removal which did not increase with exercise was commented upon but no reason could be given for it. She died on the seventh postoperative day. Our interpretation is that there was unrecognized right ventricular damage of which the only evidence was this low oxygen removal which did not increase with exercise.

The question might well be asked: what does a normal resting oxygen removal indicate in the presence of obvious parenchymal disease? Subject T. H., a 26-year-old Negro woman with tuberculosis, had a resting removal of 44 ml. which, however, decreased to 33 ml. on exercise. Our preoperative report was that she would not go through surgery due to right ventricular

---

Received for publication May 9, 1956.

*Surgeon and Director of the Cardio-Pulmonary Laboratory of the City of New York, Queens Hospital Centre, New York, N. Y.

**Resident in Anaesthesia, New York University-Bellevue Medical Centre, New York, N. Y.

still drain into the pulmonary veins.[1] Pulmonary artery shunts to the bronchial veins also develop, but these too, have the same ultimate venous outflow. It should be remembered that the pulmonary artery pressure does not increase with exercise *until the cardiac output is three times normal*, such is the flexibility of the pulmonary vascular bed.[3,4] The pulmonary artery pressure will rise when the output is twice normal if one lobe is obstructed by malignant gland pressure, or one and one half times normal if one main pulmonary artery is obstructed. It follows then, that on exertion there will be an increase in pressure (which varies with the degree of obstruction) to push by the obstruction and so tend to increase the oxygen uptake and removal of the involved area, while if the obstruction is fixed by actual invasion there can be no increase in oxygen uptake or removal by the involved lung. This point is brought out because of its bearing on the operability of a malignant lesion.

One further point is of extreme interest to the surgeon. The problem as to whether a "pulmonary malignant lesion" is primary or secondary in the lung in the presence of a negative bronchoscopy (with a doubtful Papanicolaou and a negative physical examination) can sometimes be decided one way or the other by the oxygen removal rate. If the removal is low it points to a primary lesion with large artery obstruction, if the removal is normal and more especially if it increases with exercise, the lesion is metastatic and involves *more distal finer terminal vessels by the peculiar virtue of the pathology of* metastatic spread which does not affect oxygen removal because the large pulmonary vessels are free.

Subject W. F., a 71 year old man, was admitted to the thoracic service with fluid in the right chest. This was tapped and a hydropneumothorax developed with a radiolucency in the right upper lobe and a partial atelectasis. Fluid block sections showed cells suggestive of malignancy. Supraclavicular nodes were enlarged and a negative biopsy obtained. Bronchoscopy was negative. Bone series and all other studies were completely negative for other sources of malignancy. The oxygen consumption and removal at rest was 53 ml. per liter which increased to 80 on exercise. The pathology report, four months later, was for carcinoma of the tail of the pancreas with spread to liver, lung, peritoneum, and pleura.

Subject B. A. is *another example of a patient who had a nephrectomy for* carcinoma seven years previously. He developed a right lower pulmonary malignant lesion and came to our thoracic unit with fluid as well on the right side. The oxygen removal was 29 ml. at rest and did not decrease on exercise. The absence of a fall on exercise is indicative of a metastatic type of pulmonary malignancy.

Another point of surgical importance should now be considered. If the problem lesion in the chest is benign, and most of them are problems until thoracotomy, (cystic, fibromatous, granulomatous, etc.), then they do not obstruct major vessels and the oxygen removal is relatively normal. What is more important is that the $O_2$ removal increases with exercise. If the oxygen removal is normal and especially if it increases with exercise one can state further that even if the lesion is malignant it is operable because it is of very limited extent.

damage (unrecognized). However, her disease was primarily confined to one side and a pneumonectomy was carried out. She left the operating table with a tachycardia under $O_2$ of 150 beats per minute which persisted until her death some weeks later. The post-mortem report stated that the "heart is minimally enlarged as a result of minimal hypertrophy and dilatation of the right ventricle and right ventricular outflow tract; all other chambers were normal."

Due to an increased catabolism, her oxygen demands at rest were increased by 150 per cent (294 ml. per minute). This resulted in a relatively increased perfusion through the remaining healthy tissue which more than offset the effects of a diminished perfusion through diseased tissue. The one effect offset the other and a normal $O_2$ removal value was obtained. On mild exercise, even though the consumption increased to 460 ml. per minute, the removal rate decreased to 33 ml. per liter. This showed that she was at the end of her compensating ability and the right ventricle was unable to augment its output proportionately.

Obstruction of the pulmonary artery or of a large branch from any cause, resulting in ventilation of an unperfused area of lung, lowers the oxygen removal rate.

Subject V. L. shows the physiologic effects of accidental severance of the main right pulmonary artery during lobectomy. An attempt was made at re-anastomosis. The oxygen consumption and removal at rest and exercise point to severe pulmonary vascular deficiency on exertion rather than ventilatory insufficiency since she was able to ventilate at the rate of 26 L. per minute on exercise.

Pulmonary embolism and/or thrombosis for the same reason results in a low oxygen removal.

Subject R. K., a 30-year-old woman, had had a surgical procedure two years prior to study wherein she developed a pulmonary embolus. The oxygen removal at rest and exercise was 37 and 22 ml. per liter, respectively. The oxygen consumption was 276 at rest and 369 ml. per minute on mild exercise. Vital capacity and maximum breathing capacity were 84 per cent and 129 per cent of normal, in that order. This patient showed a partial residual vascular impairment. There were no other clinical findings such as pulmonary stenosis, right ventricular incompetence, etc., which also result in low oxygen removal rates.

One of the most important surgical causes of a decreased $O_2$ removal on exercise is the extrinsic pressure and/or invasion by hilar malignant node involvement of the pulmonary artery, vein, or its main branches.

Subjects M. W. and L. H. illustrate this phenomenon. At operation, all these cases were proved, personally by the author, to have extrinsic or intrinsic vascular obstruction. Both patients had preoperative bronchospirometric studies showing diminished oxygen uptake on the side of the lesion. The oxygen removal of Subject M. W. was 31 at rest and 19 on exercise and of Subject L. H. it was 38 and 15 ml. per liter.

The validity of the postulate is not affected by the fact that the bronchial arteries form bronchopulmonary anastomoses under pathologic conditions and take up a very considerable amount of $O_2$ if there is arterial unsaturation but

If there is no arterial unsaturation it should be pointed out that a nonventilated lung, or portion thereof, automatically becomes nonperfused (the blood flow ceases to a nonfunctioning area of lung)[1] The converse of course does not follow If there is sufficient parenchymal disease (and there must be enough to show up on a flat x-ray film) this, per se, will decrease the perfusion of the involved areas If there is a corresponding amount of bronchial obstruction to ventilation or impairment through loss of mobility or flexibility of the involved parenchyma then there will be no change in the oxygen removal (both factors being affected equally) If, on the other hand, there is less loss of ventilation relative to the decrease in perfusion, then there will be a decrease in the oxygen removal

Subject G R illustrates the point just discussed His main disease (tuberculosis) involved the left upper lobe with some also in the right upper lobe Angiocardiography showed an abrupt cessation of dye at the entrance to the left upper lobe and a partial obstruction to its flow to the right upper lobe at three and four seconds, as is so common in angiocardiography films, there was some overexposure but nevertheless there was no question about it The oxygen removal was 19 ml per liter at rest and 38 on exercise This means that there was, at rest, ventilation of the nonperfused upper lobes On exercise, he was able to increase the right ventricle output to transport 527 ml of oxygen per minute and it followed that there were enough patent pulmonary arteries coursing into ventilating parenchyma to perform this function The conclusion to be drawn from the pulmonary function study alone is that since the oxygen removal showed an even better than normal percentage increase with exercise the functioning portion of the total lung parenchyma was of normal function The problem then was how much was normal lung tissue from a physiologic standpoint, as well as anatomically The resting oxygen removal was one-half of normal, therefore one half of the parenchyma was normal from both points of view Since it had now been shown that one half of the total lung tissue was of normal function, and thus was ample to sustain a satisfactory existence, the only remaining question was whether this physiologically competent lung parenchyma was situated in such a manner that it could be preserved The x rays and angiocardiograms, in addition to the function studies, confirmed that it could be preserved There were about 5 L of ventilation at rest per minute in the upper two lobes which were useless, and would be eliminated by their removal On exercise the functionless upper lobe ventilation would also be done away with The pulmonary artery pressures would be no higher after surgery than before because they were not perfused before This patient will if anything be better from a physiologic point of view after bilateral upper lobectomy

Last, the pulmonary studies pertinent to this communication of a few surgical cardiac patients are presented in Table I These all show a diminished oxygen removal which may or may not increase with exercise, depending primarily on the myocardial reserve (vide infra)

Many of these patients (and we are dealing primarily with mitral stenosis cases here) are hyperventilating constantly due to visceroceptive reflexes arising

Subject R. J., a 58-year-old man, had a large left upper lobe lesion as well as tuberculosis. The oxygen consumption at rest and exercise was 310 and 710 ml. per minute. The oxygen removals were 42 and 40, respectively. The preoperative cardiopulmonary report was "patient fit for resection. Studies suggest that this subject may have a resectable lesion if it is primarily pulmonary." Bronchoscopy was negative and the problem was to decide whether the lesion was a metastatic one (the appearance was suggestive of such) or, if primary, was it resectable? At operation, it was easily removed by an extrapleural resection. The pathology report was bronchogenic carcinoma.

In primary lesions of the mediastinum, the surgeon may infer, if the oxygen removal increases on exercise or remains the same (does not decrease) that the lesion is circumscribed or noninvasive such as would be the case in the lymphomas, cysts, neurogenic tumors, etc., whereas malignant lesions of comparable size cause a decrease in the oxygen removal on exercise.

Subject B. A., a 59-year-old man, had a tumor projecting from the mid-mediastinum and pericardium. The oxygen removal was 26 and 45 ml. per liter with an oxygen consumption of 204 and 538 ml. per minute at rest and exercise, respectively. At operation there was a tumor with a fan-shaped pedicle adherent to the pericardium in the mid-mediastinum with the phrenic nerve adherent to its posterolateral surface. The pathology report was benign teratoma.

Subject R. E., a 16-year-old girl, had a tumor mass in the mid-mediastinum projecting into the left chest. No definite preoperative diagnosis could be made. The oxygen consumption increased on moderate exercise from 170 to 415 ml. per minute. The oxygen removal did not fall on exercise. A 12 by 8 by 8 cm. benign ganglioneuroma "attached to retropleural and mediastinal ssues over a rather wide base and to the sixth intercostal nerve and sympathetic nglia" was removed. There was "pressure erosion of the mid-dorsal vertebral odies" and the mass was in the "left posterior quarter over the left hilum." ecovery was uneventful.

The effect of severe mediastinal shift and distortion on the pulmonary essels is exemplified by Subject R. B. He had a right-sided segmental resecon with a thoracoplasty as well as bilateral bronchiectasis. The oxygen reoval was only 22 ml. at rest and 15 on exercise. A ventilation rate of 15 L. er minute on exercise demonstrated that he could ventilate and it was not erely dead space ventilation since there were neither localized nor generalized nphysema nor cystic areas. In certain cases, surgical means of correcting this severe physiologic impairment might be indicated.

A second example is T. D., a 44-year-old man, who had extreme tuberculosis retraction of the right upper lobe with mediastinal shift and normal remaining lungs. The oxygen removal went down from 28 to 19 ml. per liter on exercise with an oxygen consumption increment from 224 to 392 ml. per minute. Angiocardiography later showed a displaced superior vena cava and partial obstruction of the vessels on the right confirming the lung function findings. Lobectomy will improve this patient's vascular hemodynamics.

Extreme shift of the mediastinum with tension and deflection of the vena cava or pulmonary arteries also results in a diminished oxygen removal which decreases still further with exercise. The cause is usually obvious however, e.g., large aneurysm, thoracoplasty, and localized pulmonary contracting fibrosis. The inflexibility of the causative agent results in this phenomena.

Parenchymal disease affects perfusion and ventilation proportionately so that the oxygen removal does not change with exertion even if it is somewhat decreased at rest.

Last, cardiac patients with valvular disease have a diminished oxygen removal at rest. The change with exertion depends upon whether they are in low or high output failure, if in failure, if not then it depends upon the myocardial status, right or left, or both.

The test is very easily done and when interpreted to its full extent is very helpful in diagnosis and as part of a study to assess cardiopulmonary function since it depends upon both. Psychogenic hyperventilation has been no problem with us.[10]

## SUMMARY

The cardiopulmonary studies of a variety of surgical subjects with particular reference to the oxygen removal rate, are presented.

The value in diagnosis is pointed out with confirmation of its interpretation.

The oxygen removal is diminished at rest and decreases still further on exercise in carcinoma with "inoperable mediastinal spread."

The oxygen removal does not decrease with exercise in benign lesions and metastatic peripheral pulmonary lesions.

Valvular disease and right and/or left sided myocardial disease also cause a decreased oxygen removal at rest. The reaction to exercise varies with the status of the myocardium and hemodynamics. The oxygen removal is a good index of the myocardial reserve.

## REFERENCES

1. Shedd, D. P., Alley, R. D., and Lindskog, G. E.: Observations on the Hemodynamics of Bronchial Pulmonary Vascular Communications, J. Thoracic Surg. 22: 537, 1951.
2. Hickam, J. B., and Cargill, W. H.: The Effect of Exercise on Cardiac Output and Pulmonary Arterial Pressure in Normal Persons and Persons With Cardiovascular Disease and Pulmonary Emphysema, J. Clin. Invest. 27: 10, 1948.
3. Riley, R. L., Himmelstein, A., Motley, H. L., Weiner, H. M., and Cournand, A.: Studies of Pulmonary Circulation at Rest and During Exercise in Normal Individuals and in Patients With Chronic Pulmonary Disease, Am. J. Physiol. 152: 372, 1948.
4. Cournand, A., Riley, R. L., Himmelstein, A., and Austrian, R.: Pulmonary Circulation and Alveolar Ventilation—Perfusion Relationships After Pneumonectomy, J. Thoracic Surg. 19: 80, 1940.
5. Pitts, R. F.: Textbook of Physiology, ed. 16, Philadelphia, 1949, W. B. Saunders Company, chapt. 42.
6. Personal communication of Dr. K. Roberts, Memorial Hospital, New York, N. Y.
7. Bjurstedt, A. G. H.: Interaction of Centrogenic and Chemoreflex Control of Breathing During Oxygen Deficiency at Rest, Acta physiol. Scandinav. (Suppl. 38) 12: 1, 1946.
8. Cournand, A.: Cardio-Pulmonary Function in Chronic Lung Disease, Harvey Lecture Series (1950-51), vol. 43, Springfield, Ill., 1952, Charles C Thomas, Publisher, pp. 68-97.
9. Bing, R. J.: Coronary Circulation in Health and Disease as Studied by Coronary Sinus Catheterization, Bull. New York Acad. Med. 27: 408-424, July, 1951.
10. Gaensler, E. A.: Medical Progress, Clinic Pulmonary Physiology, New England J. Med. Nos. 5, 6, and 7: 252, 1955.

from the chronically stimulated alveolar capillary wall as a result of an increased pulmonary capillary pressure.[5] This in turn "blows off $CO_2$," which results in an increased sensitivity of the respiratory center in the floor of the fourth ventricle, because it results in the kidney releasing $HCO_3$ to lower the raised pH back to normal.* The renal loss of excess $HCO_3$ lowers the total ionic plasma content which increases the respiratory center sensitivity.[7, 8] Any slight increase in the intracellular $CO_2$ of this oversensitized respiratory center due to exercise will augment still more the respiratory rate. Since the resting cardiac output usually is a low normal or even less there is a high arteriovenous oxygen difference (low venous oxygen content) in the range of 7 to 10 volumes per cent. On exercise, due to the valvular disease, myocardial damage or lack of reserve, the output cannot be increased, and even may decrease, which, coupled with a resting low venous oxygen content, results in an inability of the hemodynamic mechanism to increase significantly the total oxygen uptake from the lungs. Thus, we have in exercise an increase in ventilation accompanied by a small or no increase in oxygen uptake. This shows itself as an oxygen removal on exertion equal to or less than that at rest.

The resting oxygen removal is low because of the aforementioned augmented visceroceptive stimulation causing hyperventilation in the face of a normal oxygen consumption. In cardiac disease, there is a normal total oxygen consumption and in fact the oxygen consumption per kilogram of myocardium is also normal (the efficiency, however, is down); as a matter of fact, even in the cardiac hypertrophy of hypertension there is no increase in oxygen consumption per kilogram of heart muscle mass.[9]

## DISCUSSION

Right ventricular damage which may be unrecognized per se shows itself in pulmonary function studies as a diminished oxygen removal. This diminution is even more marked on exercise.

The first and second patients were used to illustrate this (L. L. and T. H.). The heart can neither maintain nor increase its oxygen removal rate on exercise even with a normal left ventricle.

Malignant lesions by virtue of intraluminal neoplastic invasion or unyielding extraluminal compression result in a decreased oxygen removal at rest which further decreases with exertion. Accidental ligation will, of course, produce a similar picture.

Pulmonary arterial obstruction from benign lesions do not cause a further decrease in the oxygen removal on exercise because the increased ventilatory movement on exertion together with the increased cardiac output is able to effectively by-pass this "benign obstruction" and the oxygen removal increases with exercise. Secondary pulmonary metastatic lesions act similarly through the peculiar pathology of their method of spread. They first grow near small vessels and so do not obstruct much of the blood flow.

---

*There is recent work which may modify the present usually accepted description of a "compensated respiratory acidosis" as one in which renal NA+ and $HCO_3-$ retention offsets an increased $PCO_2$ blood level.[6]

(M.M.N.) was chosen for our initial experiments because of its mechanical simplicity, ruggedness, ease of insertion and fixation in the mitral annulus, and ease of manufacture in our shop.*

The technique finally evolved was this: The animal's circulation was connected to the pump-oxygenator. Part of the blood flow at this time still passed through its own heart and lungs In 23 dogs, ventricular fibrillation was initiated with a shock of 6 volts 60 cycle AC through two closely spaced electrodes applied to the ventricle. Fibrillation occurred spontaneously in 2 animals A few seconds later an incision, 3 cm. long, was made from the midpoint of the left auricular appendage to a point past the pericardial reflection on the lett inferior pulmonary vein. A small vein retractor was inserted to retract the auricle anteriorly to expose the mitral valve, traction on the auricular appendage was also essential for good exposure. Blood in the field was removed by continuous suction to a sterile bottle tor possible later re-introduction into the circulation. A continuous purse-string suture of No. 2 braided silk was introduced in such manner that suture bites passed through the elastic tissue of the mitral annulus. Chordae tendineae to both mitral leaflets were then divided and, occasionally, portions of the valves were excised to remove any obstructions to the prosthesis entering the left ventricle. The valve was slipped into position and the heavy silk purse-string suture was tied into the groove around the base of the valve. The auricle was then allowed to fill with blood and all air was displaced from the left ventricle. The auricular incision was closed with two layers of continuous, Atraumatic No 5-0 silk.

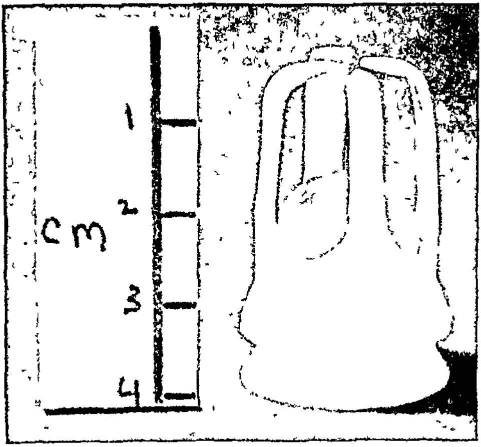


Fig 1—Mitral valve prosthesis

---

*We are indebted to Mr Ludwig Quabeck for the precision work on the pump-oxygenator and on the valves

# A PROSTHESIS TO REPLACE THE MITRAL VALVE

M. C. Kernan, M.D., M. M. Newman, M.D., B. S. Levowitz, M.D.,
J. H. Stuckey, M.D., and C. Dennis, M.D., With the Technical Assistance
of Alva Riddick and Herbert Lauritzen
Brooklyn, N. Y.

At present, there is no good surgical treatment for severe mitral insufficiency, especially for that secondary to severe calcification of the mitral leaflets or destruction of chordae tendineae. Experimental work on dogs has shown that the mitral valve can be replaced completely by a plastic prosthesis. To our knowledge, the only previous artificial mitral valve which has permitted prolonged survival is that reported by Denton.[1]

## METHODS

Dogs weighing 17 to 25 Kg. were anesthetized with intravenous pentobarbital. A cuffed, intratracheal tube was inserted and ventilation begun with the Jefferson Respirator* with 100 per cent $O_2$. The left chest was entered through the bed of the fifth rib. The left subclavian artery was isolated. The pericardium was opened transversely from the reflection over the left pulmonary veins to well over on the right side, the phrenic nerve being dissected free and retracted laterally.

Heparin was given intravenously at this point. Prior to perfusion the heparin level in the blood used to prime the extracorporeal circuit averaged 60 mcg. per milliliter (heparin†) as measured by titration with protamine sulfate (1 per cent solution N. F.‡). This level in the priming blood resulted from the addition of 25 mg. of heparin to each liter of blood drawn.

The subclavian artery was ligated where it gave off the vertebral artery and was divided. A No. 14 Fr. polyvinyl catheter was inserted through the proximal stump to just within the aorta or slightly into descending aorta. A No. 28 Fr. catheter was secured in the right atrium by a purse-string suture in the atrial appendage in the last 16 dogs instead of encannulating each vena cava. The pump-oxygenator was attached to the arterial and venous channels. Technical details of the artificial heart-lung have been described previously.[3, 4]

The valve is shown in Fig. 1. It is a Teflon shell, housing a solid methyl methacrylate sphere,§ 15.5 mm. in diameter. This design for a ball valve

---

From the Department of Surgery, State University of New York College of Medicine.

This work was supported by grants from the U. S. Public Health Service, The Life Insurance Medical Research Fund, and the State University of New York.

Received for publication May 11, 1956.

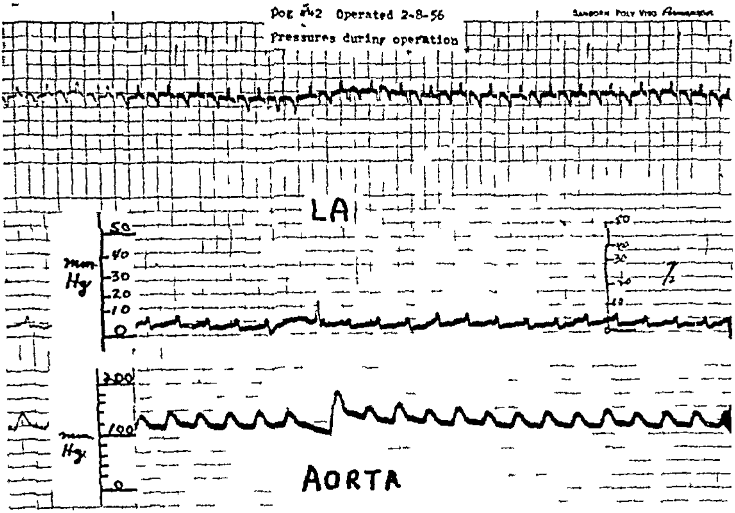
*Air Shields, Inc., Hatboro, Pa.

†Supplied by Connaught Laboratories.

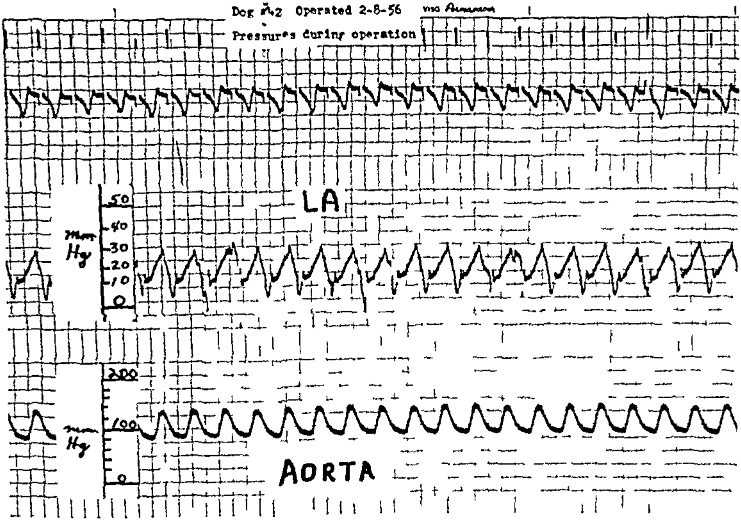
‡Supplied by Eli Lilly Company.

§Lucite spheres obtained from Ace Plastic Company, 91-30 Van Wyck Expressway, Jamaica, N. Y.

In two animals, nylon balls were used.



*A*



*B*

Fig 2—Pressure tracings in the left auricle taken during operation before and after insertion of the prosthesis Dog No 42 *A* Before insertion *B* After insertion (*Fig 2 continued on pp 702 and 703*)

The heart was massaged a few times to relieve any distention of the right ventricle. Defibrillation was usually accomplished with one or two shocks of about 2 amperes of 0.1 second duration. A loud systolic click marked onset of strong left ventricular contraction.

The chest was drained through tubes in the right and left sixth interspaces. The periosteal bed and muscles were closed in layers with interrupted silk.

## RESULTS

The procedure described was performed on 25 dogs. The first 7 were considered as pilot studies. Three died during operation and 4 within the first six hours, postoperatively. In 4 of these dogs, the mitral valve was approached through the left auricle and the prosthesis fixed with interrupted sutures. A ventricular incision was used in 3 dogs with fixation by interrupted sutures in 2 and a circumferential suture in one. All 3 dogs with ventricular incisions died in one and one-half hours or less.

The remaining 18 dogs were all operated upon by essentially the same technique: a left auricular approach and a purse-string suture around the annulus for fixation. There were five fatalities during the first 48 hours postoperatively, 12 dogs lived 48 hours to 3 weeks, and one died 4 months after insertion of the prosthesis (Table I).

Intra-auricular pressures, recorded before the auricle was opened and after the valve had been placed, show the absence of regurgitation (Fig. 2 and Table II). However, a minimal amount of opacification is seen on motion picture cardiograms in which dye was injected directly into the left ventricle. The tracings do show moderately elevated intra-auricular pressures during auricular systole. When tested outside the heart, the valve seemed to produce minimal obstruction to flow: a 10 cm. head of pressure produced a flow of more than 20 L. per minute.

TABLE I. SURVIVAL TIMES AND CAUSES OF DEATH PLASTIC MITRAL VALVE

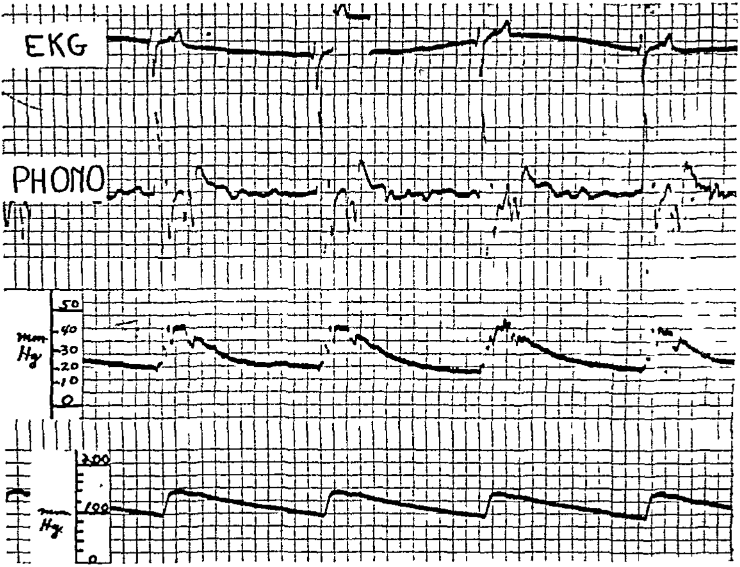
| CAUSES OF DEATH | NUMBER OF DOGS | DURATION OF SURVIVAL |
|---|---|---|
| Technical error | 3 | Less than 48 hours |
| Hemorrhage | 2 | Less than 48 hours |
| Thrombosis on purse-string suture and fatal emboli | 5 | 2 to 21 days |
| Displacement of valve (nonfatal emboli also present) | 5 | 2 to 21 days |
| Extrusion of ball | 2 | 4 days and 4 months |
| Occlusion of valve by thrombus | 1 | 10 days |

TABLE II. AURICULAR PRESSURES

| DOG NUMBER | BEFORE (MM. HG) | AFTER (MM. HG) |
|---|---|---|
| 544 | 7/0 | 20/10 |
| 27 | 5/0 | 18/3-5 |
| 32 | 8/0-2 | 20-22/2-4 |
| 42 | 7/2 | 30/5-15 |
| *Wedge Pressures* | | |
| 591 | | 18/0 |
| 587 | | 20/0 |

a double suture of silk or pericardium at the aortic-auricular junction, passing through the valve and out the anterior and posterior walls of the atrium.[9]

Bailey and his associates[10, 11] have reported use of a noose of pericardium inserted through the ventricle and across the posterior commissure. Also pericardial strips have been used by this group as artificial chordae tendineae for placement between the valve cusp and ventricular wall.



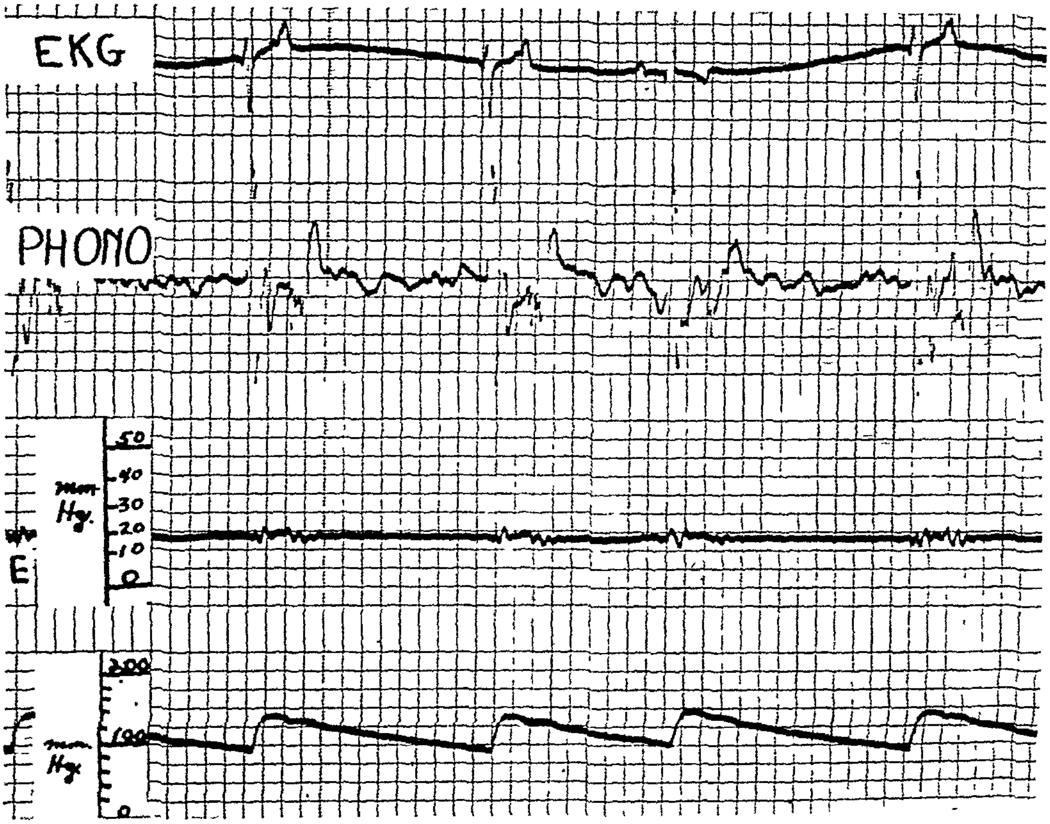
*D*

Fig 2 (Cont'd) —*D*, Wedge pressure taken one month postoperatively Dog No 59.

Any procedure modifying the valve structure or ring is applicable only where the structures are pliable and not calcified or where the loss or distortion of tissue is not too great. Correction of a defect requires additional material which is usually placed beneath the valve. Such defects usually occur in the posterior commissure or central portion of the valve It would be most desirable to have the prosthesis at right angles to the defect to obtain optimal function and to avoid the papillary muscles and chordae However, this is impossible since the axis would traverse the interventricular septum and aorta. This method, therefore, has great disadvantages no matter what material is used.[12]

Although not fixed in an ideal position, autogenous grafts such as inverted veins, tendons, free and pedicled strips of pericardium have all met with initial success when used as transventricular slings. It became clear that failure of these materials to retain their original form was due to chronic inflammatory

The control clotting time averaged 12 minutes while postperfusion it was 15 minutes. The control $pCO_2$ was 39 mm. Hg average and postperfusion was 23 mm. Hg. The mean perfusion arterial pressure was 68 mm. Hg. The average arterial saturation was 96 per cent and flows averaged 39 c.c. per kilogram per minute.



C.

Fig. 2 (Cont'd).—*C.* Pulmonary artery. Dog No 59

## DISCUSSION

Attempts to correct mitral insufficiency can be divided roughly into two groups: reconstructive procedures on the valve proper, and those operations in which an additional substance is used to support or replace the distorted valve.

The first recorded plastic procedure to correct mitral insufficiency was performed in dogs by Alexis Carrel.[5] He performed a partial resection of the wall of the left ventricle just below the coronary artery with a plication of the defect which reduced the size of the mitral annulus. This dog survived—for what period of time is not known. Successful experimental work of a similar type was reported by Hurwitt, Hoffert, and Ferreira.[6]

Recently, Davila, Mattson, O'Neill and Glover[7, 8] have shown the results of decreasing a dilated atrioventricular ring by a purse-string suture placed externally. Another method of decreasing the diameter of the annulus is to place

The portion of the valve projecting into the left ventricle undoubtedly injures the ventricular wall and septum during each ventricular contraction In some animals, we have seen small subendocardial hematomas and scarring The desirability of eliminating this constant source of trauma is recognized, and will be considered in future designs

The purse-string suture securing the valve has not been satisfactory for long-term survival except in one dog, although it has allowed opportunity for testing other characteristics of the prosthesis Various other fixation techniques are being tried

## CONCLUSIONS

1 A prosthesis consisting of a Teflon casing and Lucite ball has been developed to replace the mitral valve in dogs

2 Using a pump oxygenator and ventricular fibrillator, we have successfully inserted the valve in 18 dogs in which mitral regurgitation had been created by severing chordae tendineae and by partial amputation of mitral leaflets

3 Survival times have varied from a few hours to 4 months The longer term survivors have not appeared to suffer exercise intolerance

4 The related problems of fixation and thrombus formation at the zone of fixation must be further investigated before this mechanically satisfactory ball valve can be adapted to clinical use

## REFERENCES

1 Denton, G R A Plastic Prosthesis Without Moving Parts for the A V Valves, Proc Surg Forum, Clinical Congress, College Surgeons, 1950, Philadelphia, 1951, W B Saunders Company, p 239 45
2 Denton, G R, Seymour, T, and Wiggers, H A Follow Up Report on the Development of a Plastic Prosthesis for the A V Valve, Proc Surg Forum 1953, Clinical Congress Am Coll Surgeons, Philadelphia, 1954, W B Saunders Company, p 83 7
3 Dennis, C, Spreng, D J, Nelson, G E, Karlson, K E, Nelson, R M, Thomas J V, Eder, W P, and Varco, R L Development of a Pump Oxygenator to Replace the Heart and Lungs, an Apparatus Applicable to Human Patients and Application to One Case Ann Surg 134 709 1951
4 Newman, M M, Stuckey, J H, Levowitz B S, Young, L A, Dennis, C, Fries, C C, Gorayeb, E, Zuhdi, M, Karlson, K E, Adler, S, and Gliedman, M Complete and Partial Perfusion of Animal and Human Subjects With the Pump Oxygenator, Surgery 38 30, 1955
5 Carrel, A On the Experimental Surgery of the Heart and Thoracic Aorta, Ann Surg *52 83, 1910*
6 Hurwitt, E S, Hoffert, P W, and Ferreira, R The Experimental Production and Correction of Mitral Insufficiency, Surgery 37 15, 1955
7 Davila, J C, Mattson, W W, Jr, O'Neill, T J E, and Glover, R P A Method for the Surgical Correction of Mitral Insufficiency 1) Preliminary Considerations, Surg, Gynec & Obst 98 407, 1954
8 Davila, J S, Glover, R P, Trout, R D, Mansure, F S, Wood, N E, Janton, O H, and Iaia, B D Circumferential Suture of the Mitral Ring, J THORACIC SURG 30 531, 1955
9 Kay, E B, and Cross, F S Surgical Treatment of Mitral Insufficiency, J THORACIC SURG 29 618, 1954
10 Bailey, C P, Jamison, W L, Bakst, A A, Bolton, H E, Nichols, H T, and Meinhardt, W G The Surgical Correction of Mitral Insufficiency by Use of Pericardial Grafts, J THORACIC SURG 28 551, 1954
11 Bailey, C P, O'Neill, T J E, Glover, R P et al Surgical Repair of Mitral Insufficiency By Use of Pericardial Grafts, Dis Chest 19 125, 1951
12 Brock, R C The Surgical and Pathological Anatomy of the Mitral Valve, Brit Heart J 14 489, 1952

changes which were thought to be a result of the avascularity of the transplanted tissue.[13-16] Attempts were made to remedy this by inverting a piece of vein over internal mammary artery, but without much success.[17] A nylon ball within a tube of pericardium, used as a sling, failed ultimately for the same reason: avascular changes which would in all probability have led to embolization of the ball.[18]

The auricular appendage has been used to fashion an additional valve by invaginating it or anastomosing it to the ventricular wall, but again long-term results have been unsatisfactory.[1, 19, 20]

A somewhat different approach has been the bottle-shaped Lucite baffle used by Harken,[21] the Ivalon baffle of Johns and Blalock,[22] and the Lucite ball attached to the posterior wall of the left ventricle.[21, 23, 24] These prostheses are at best limited in application and unpredictable in outcome.

Still another type of replacement, made of spring steel ("Elgiloy") and nylon, has been used experimentally with little success by Jordan and Wibble.[25] Inserted through a trocar in the left ventricular wall, the nylon-covered steel springs apart at a 45 degree angle, bridging a defect in the posterior commissure with each systolic thrust.

Hufnagel[26] has shown that a plastic prosthesis in the descending aorta can successfully substitute for a severely insufficient aortic valve. To replace the mitral valve with an intracardiac prosthesis is a more formidable undertaking and yet seems the most universally applicable solution. Criteria which such a prosthesis must meet are: the valve must (1) be mechanically efficient—there must be no stenosis or regurgitation, (2) be of a stable and durable material, (3) interfere little or not at all with ventricular contraction, (4) be well tolerated by tissues, (5) be simply and securely anchored, (6) be free of clotting, and (7) be adaptable to a wide variety of clinical situations.

In 1950, Denton, Mastriani, Brown, and Wiggers reported the successful replacement of the mitral valve by a rigid polyethylene prosthesis sutured into the mitral ring.[27] This was rounded on its ventricular end with either a straight tubular opening or a side opening which was closed by ventricular contraction. The longest survivors lived four to five months. Denton[1] has since reported extensive experimental work with this type of valve. Anchoring the prosthesis proved to be so technically difficult and displacement of the valve was so common, that this approach to mitral valve replacement has been abandoned.[2]

The Teflon and Lucite valve developed in our laboratory has proved to be a mechanically efficient, rugged replacement. Teflon was selected for the casing because it possessed high tensile strength, chemical inertness, and low wettability. Polished Lucite spheres were used because they are unlikely to promote clotting.

Following the extensive work of Senning[30] and Glenn and Sewell,[31] ventricular fibrillation has been shown to be a completely satisfactory way of preventing air embolism, which is a particularly great hazard in surgery on the left side of the heart. Another advantage of fibrillation is that of better visualization and easier manipulation in a still heart. We have had no difficulty in defibrillating a heart which has been well oxygenated during adequate perfusion.

# The Journal of
# Thoracic Surgery

VOL. 33 JUNE, 1957 No. 6

## Original Communications

# A STUDY OF THE CARDIOVASCULAR CHANGES DURING COOLING AND REWARMING IN HUMAN SUBJECTS UNDERGOING TOTAL CIRCULATORY OCCLUSION

EMIL BLAIR, M.D.,* R. REED AUSTIN, M.D.,* S. GILBERT BLOUNT, JR., M.D., AND
HENRY SWAN, M.D.
DENVER, COLO.

ALTHOUGH therapeutic successes with general hypothermia have been reported for slowing disease processes and for relief of pain,[6, 11] it is only recently that its important role has been recognized in cardiovascular surgery.[2, 3, 4, 7, 12] Reduction of metabolism by hypothermia allows total occlusion of the circulation for a sufficient period of time to permit repair of certain types of vascular and cardiac defects. Our clinical experience with 100 patients undergoing open cardiotomy has indicated that total circulatory occlusion of 8 minutes at a body temperature not lower than 28° C. is relatively safe and affords adequate time to repair interatrial septal defects, pulmonary valvular stenosis, and pulmonary infundibular stenosis.

While numerous studies of cardiovascular physiologic changes occurring during hypothermia in animals have been reported, and several studies of the effect of cold on unanesthetized human beings, there have been relatively few dealing with human patients during hypothermia. With specific reference to the cardiovascular system, Talbott[13] found that the pulse and blood pressure initially rose upon inducing hypothermia in man, and that both then gradually fell as the temperature reached 28° to 32° C. He observed that frequently no

---

From the Departments of Surgery and Medicine, University of Colorado School of Medicine, Denver, Colo.

Aided in part by a grant from the U. S. Public Health Service (H-1559 C), and in part by a grant from The American Heart Association.

Received for publication May 24, 1956.

*U. S. Public Health Service Post-Doctorate Research Fellow.

13. Murray, G., Wilkinson, F. R., and MacKenzie, R.: Reconstruction of the Valves of the Heart, Canad. M. A. J. 38: 317, 1938.
14. Templeton, J. Y., and Gibbon, J. H.: Experimental Reconstruction of Cardiac Valves by Venous and Pericardial Grafts, Ann. Surg. 129: 161, 1949.
15. Henderson, A. R., and Law, C. L.: The Surgical Treatment of Mitral Insufficiency, Surgery 33: 858, 1953.
16. Moore, T. C., and Shumacker, H. B., Jr.: Unsuitability of Transventricular Autogenous Slings for Diminishing Vascular Insufficiency, Surgery 33: 173, 1953.
17. Glenn, W. L., and Turk, L. N.: The Surgical Treatment of Mitral Insufficiency; the Fate of a Vascularized Transchamber Intracardiac Graft, Ann. Surg. 141: 510, 1954.
18. Carter, M. G., Gould, J. M., and Mann, B. F.: Surgical Treatment of Mitral Insufficiency, J. Thoracic Surg. 25: 574, 1953.
19. Rappaport, A., Murray, J. F., and Davies, L. S.: Atrioventricular Anastomosis; an Additional Valve, Proc. Soc. Exper. Biol. & Med. 69: 345, 1948.
20. Hayward, J.: A New Operation for Mitral Regurgitation, Australian & New Zealand J. Surg. 23: 4, 1954.
21. Harken, D. E., Black, H., Ellis, L. B., and Dexter, L.: The Surgical Correction of Mitral Insufficiency, J. Thoracic Surg. 28: 604, 1954.
22. Johns, T. N. P., and Blalock, A.: Mitral Insufficiency: The Experimental Use of a Mobile Polyvinyl Sponge Prosthesis, Ann. Surg. 140: 333, 1954.
23. Chalnot, P., Benichoux, R., Chardot, C., and Fritsch, R.: L'Insuffiance mitrale experimentale et sa correction par une prosthese en matiere plastique, Arch. mal. coeur 47: 317, 1954.
24. Benichoux, R., and Chalnot, P.: A Method for the Surgical Correction of Mitral Insufficiency, J. Thoracic Surg. 30: 148, 1955.
25. Jordan, P., and Wibble, J.: Spring Valve for Mitral Insufficiency, A. M. A. Arch. Surg. 71: 468, 1955.
26. Hufnagel, C. A., Harvey, W. P., Rabil, P. J., and McDermott, T. F.: Surgical Correction of Aortic Insufficiency, Surgery 35: 673, 1954.
27. Denton, G. R., Mastriani, B., Brown, C., and Wiggers, H. C.: A Physiological Effect of a Plastic Prosthesis for Mitral Valve in Dogs, Fed. Proc. 9: 31, 1950.
28. Hirschbeck, J. S.: Delayed Blood Coagulation Time in Methyl Methacrylate Vessels, Proc. Soc. Exper. Biol. & Med. 47: 311, 1941.
29. Hufnagel, C. A.: Permanent Intubation of the Thoracic Aorta, Arch. Surg. 54: 382, 1947.
30. Senning, A.: Ventricular Fibrillation During Extracorporeal Circulation, Acta chir. scandinav. (suppl.) 171: 22, 1952.
31. Glenn, W. L., and Sewell, W. H.: The Prevention of Air Embolism in Open Heart; Repair of Intra-auricular Defects, Surgery 34: 195, 1953.

## RESULTS

As already noted, the studies were done in the operating room during periods of critical surgery. Considerations directly relating to the patient's welfare, of course, took top priority. As a consequence, it was often necessary to compromise in obtaining observations. Complete data were not obtained in every patient at every phase of the study. However, sufficient data were available in the group as a whole to permit some general conclusions.

Further, it should be pointed out that the dynamics of the circulation after release of occlusion were no doubt influenced by blood loss, cardiac manipulation, and the corrective surgery.

Details relating to age of patients, together with the range of hypothermia, diagnoses, and surgical procedures, appear in Table I.

TABLE I. AGE, LOWEST TEMPERATURE AND TYPE OF OPERATIVE PROCEDURE PERFORMED

| NO. | PATIENT | AGE (YEARS) | DIAGNOSIS | TEMPERATURE (°C.) | SURGERY |
|---|---|---|---|---|---|
| 1. | P. S. | 7 | IASD | 26.0 | Closure |
| 2. | B. J. | 4 | IASD-IVSD | 28.0 | Closure |
| 3. | H. B. | 10 | IASD | 27.0 | Closure |
| 4. | S. S. | 3 | PVS | 29.0 | Valvuloplasty |
| 5. | L. B. | 3 | PVS | 31.0 | Valvuloplasty |
| 6. | H. T. | 4 | PVS | 29.0 | Valvuloplasty |
| 7. | R. B. (1) | 6 | TF | 28.0 | Valvuloplasty |
| 8. | E. C. | 8 | TF | 28.0 | Valvuloplasty |
| 9. | C. Z. | 3 | PVS | 27.0 | Valvuloplasty |
| 10. | L. H. | 9 | IPS | 27.0 | Infundibulectomy |
| 11. | J. J. | 7 | IASD | 27.0 | Closure |
| 12. | R. M. (1) | 10 | IASD | 30.0 | Closure |
| 13. | E. H. | 13 | IASD | 27.0 | Closure |
| 14. | R. B. (2) | 13 | IASD | 26.0 | Closure |
| 15. | R. M. (2) | 14 | PVS-PDA | 29.0 | Valvuloplasty; Ligation PDA |
| 16. | E. A. | 27 | IASD-PDA | 26.0 | Closure; Ligation PDA |
| 17. | T. G. | 32 | IASD | 26.0 | Closure |
| 18. | F. H. | 31 | IASD | 26.0 | Closure |
| 19. | H. H. | 35 | IASD | 26.0 | Closure |
| 20. | J. St. J. | 20 | IASD | 28.0 | Closure |
| 21. | S. P. | 17 | PVS | 28.0 | Valvuloplasty |
| 22. | V. K. | 26 | TF | 28.0 | Valvuloplasty |
| 23. | C. R. | 30 | IASD | 27.0 | Closure |

IASD=Interatrial septal defect
IVSD=Interventricular septal defect
TF=Tetralogy of Fallot
PDA=Patent ductus arteriosus
PVS=Pulmonary valvular stenosis
IPS=Infundibular pulmonary stenosis

Adequate data were obtained on 23 patients, ranging in age from 3 to 35 years. All of the patients had congenital heart disease, with most having either interatrial septal defect or pulmonary valvular stenosis. Three patients had tetralogy of Fallot, and 2 others had, in addition to the septal or valvular lesion, an associated patent ductus arteriosus. Hypothermia levels ranged from 26° to 31° C.

*Cooling.*—During the course of cooling the patient, it was noted that at varying temperature levels the sounds identifying blood pressure by the sphygmomanometer became inaudible and the pulse nonpalpable. This is referred to as "clinical disappearance" of the blood pressures (Table II). In 4 patients, the blood pressures were lost to auscultation even before the pulse or blood pressure could be obtained clinically in the cooled state. Much the same observations on pulse and blood pressure were recorded by Kossmann[6] and by Wayburn.[15]

The present study is the first to our knowledge which is concerned with cardiovascular observations made in the operating room throughout the course of intracardiac surgery in patients subjected to general hypothermia with sigmoid temperatures between 26° and 31° C. Primary interest was centered on the effect on the heart rate, blood pressure, and venous pressure under the following conditions: (1) induction and initial stage of hypothermia, (2) total circulatory occlusion, (3) release of occlusion, and (4) rewarming.

## METHOD

Details regarding the technique of immersion hypothermia and rewarming used at the University of Colorado Medical Center have been described elsewhere.[12] After premedication with Demerol and scopolamine, the patient was anesthetized with ether, and a No. 18 Cournand needle was placed into the brachial artery. A polyethylene catheter was introduced via an antecubital vein into the subclavian vein or superior vena cava. Pressures were transmitted to a carrier-type recorder. Mean pressures were either electronically integrated or computed with a compensating planimeter. Standard electrocardiogram Lead II was continuously monitored on an oscilloscope and recorded, together with the pressures. In addition, the electocardiogram was relayed to a direct writer.

Base line pressures were taken on the operating table with the patient in surgical anesthesia (third stage, first or second plane). The patient was then placed into a tub of tap water and immersed to the neck. Care was taken to level the strain gauge at the zero point of the right atrium, chosen as a point 10 cm. from the back in the adult and 6 to 8 cm. in the child. Venous pressures could not be recorded while the patient was being cooled, since the catheter was looped above the patient over the edge of the tub. The same was true for the catheter leading from the artery, but here the error introduced by correction for the difference in height between the gauge and the right atrium was not of a significant magnitude.

Ice was added to the water and shortly the temperature of the bath dropped to 5° C. or lower. Shivering was suppressed by the use of d-tubocurarine. Observations were taken every 10 minutes or for every degree fall in temperature. When the desired level was reached, the patient was lifted out of the tub and onto the operating table. During the period of preparation of the patient for surgery it was often difficult to follow the pressures. After the temperature stabilized, pressures were taken every 15 minutes or as desired by the anesthesiologist, who frequently was unable to feel the pulse or obtain a blood pressure. Continuous recording of the entire period of occlusion and the immediate phase of the release was obtained.

When the actual procedure within the heart had been accomplished, rewarming via short-wave diathermy was instituted. Pressures were again taken every 15 minutes up to 34° C., at which point the diathermy was stopped.

On cooling there occurred an increase in venous pressure from the post-anesthetic but still normothermic level, with the largest increases being observed in patients cooled to the lower temperature levels (Table III). On rewarming, 3 of 5 patients in whom adequate rewarm data were obtained had persistently elevated venous pressures above normal.

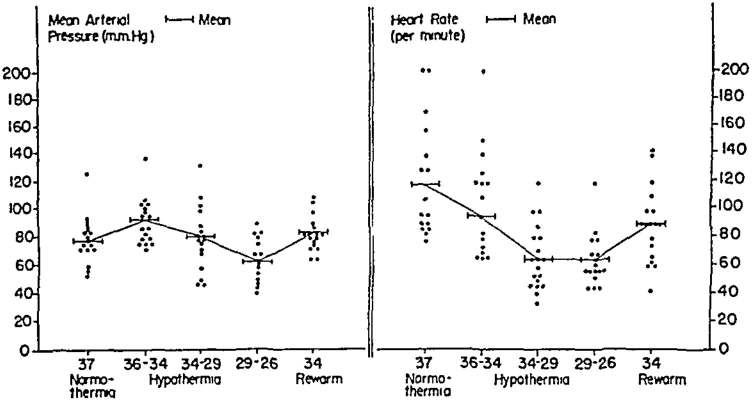


Fig 1.—Blood pressure and pulse rate changes during cooling and rewarming. As cooling proceeds there is a transient rise in blood pressure, whereas the heart rate progressively declines. Thirty minutes after lowest temperature is reached the blood pressure has further declined although the heart rate remains stable. On rewarming to 34° C. the blood pressure returns to normal, while the heart rate remains low.

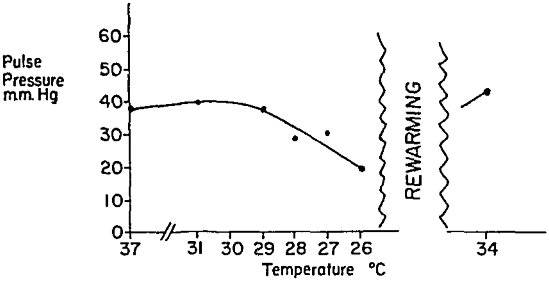


Fig 2.—Pulse pressure changes during hypothermia and rewarming. There is no change in pulse pressure until the temperature drops below 29° C. Each point represents the means of the final, stabilized temperatures.

*Total Circulatory Occlusion.*—Adequate data during total circulatory occlusion were obtained in 16 patients and are summarized in Table IV.

Immediately upon occlusion, blood pressures in all of the patients fell. Generally they were stabilized at their lowest level within one or 2 minutes, although in patients with occlusions maintained beyond 4 minutes a slight additional fall usually occurred.

temperature had dropped 1° C. In the rest of the group except one, the blood pressures could not be identified below 31° C. Direct arterial tracings at the time of the "clinical disappearance" showed the blood pressure to be quite adequate.

TABLE II. "CLINICAL DISAPPEARANCE" OF BLOOD PRESSURE AND PULSE DURING COOLING AND RETURN DURING REWARMING

| | BLOOD PRESSUBE (MM. HG) | | | | PULSE (PER MINUTE) | | | |
|---|---|---|---|---|---|---|---|---|
| PATIENT | DISAPPEARANCE TEMPERATURE °C. | ACTUAL* PRESSURE | REAPPEARANCE TEMPERATURE °C. | ACTUAL* PRESSURE | DISAPPEARANCE TEMPERATURE °C. | ACTUAL** RATE | REAPPEARANCE TEMPERATURE °C. | ACTUAL** RATE |
| P. S. | 34.0 | *95/ 62 (73)* | *35.0* | *104/66 (75)* | -- | -- | -- | -- |
| B. J. | 31.0 | 155/103 (134) | -- | -- | Palpable throughout | | | |
| H. B. | 35.8 | 96/ 64 (80) | 33.0 | (94) | 35.0 | 102 | 33.0 | 126 |
| *L. B.* | *32.9* | *107/ 56 (80)* | -- | -- | *30.0* | 83 | -- | -- |
| H. T. | 33.2 | 93/ 45 (67) | -- | -- | 31.0 | 66 | -- | -- |
| R. B. | 37.0 | (78) | 32.6 | (75) | 33.0 | 60 | -- | -- |
| E. C. | 36.5 | 120/ 80 (94) | -- | -- | 29.9 | 54 | 31.2 | 60 |
| E. A. | 27.6 | 110/ 78 (95) | 32.6 | (89) | 32.4 | 48 | 32.6 | 60 |
| T. G. | 37.0 | 120/ 86 (93) | 33.0 | 84/56 (67) | Palpable throughout | | | |
| F. H. | 36.0 | (80) | 32.6 | 135/75 (108) | 36.0 | 80 | 32.6 | 60 |
| H. H. | 32.5 | 100/ 59 (85) | -- | -- | 31.5 | 42 | 29.0 | 54 |
| *J. St. J.* | *33.0* | *91/ 61 (77)* | *32.0* | *100/59 (80)* | *33.0* | *66* | *32.0* | *54* |
| S. P. | 37.0 | 73/ 51 (56) | 30.0 | 115/60 (88) | 37.0 | 84 | 30.0 | 84 |

( ) Mean pressure.

*Direct arterial pressure.

**Obtained from electrocardiogram.

"Clinical disappearance" refers to unobtainable blood pressure and nonpalpable pulse. Note the marked variation of temperature levels when changes in both blood pressure and pulse occurred. The blood pressure and pulse at the time of "clinical disappearance" are adequate.

Pulses also were lost to palpation at varying temperatures, usually at levels of cooling lower than the blood pressure disappearance. In 2 patients, B. J., aged 3 years, and T. O., aged 32 years, the pulses were palpable throughout. Rates as determined from electrocardiogram and pulse pressures as determined from recorded contours were slow and adequate, respectively.

Upon rewarming, both vital signs "reappeared" at temperature levels lower than those at which each had disappeared.

As cooling progressed from 36° to 34° C., direct arterial pressure measurements revealed transient elevations in blood pressure, while the heart rate, on the other hand, continued in a progressive decline (Fig. 1). Following this elevation, the blood pressure began to fall to levels considerably below the normothermic levels. After the lowest temperature levels had been reached, two interesting phenomena were observed: (1) the blood pressure continued to fall a significant degree after temperature stabilization at the lowest level; (2) conversely, the heart rate did not alter appreciably during this interval of time. On the average, 30 minutes elapsed between the time of the lowest temperature stabilization to lowest blood pressure stabilization.

There was no change in pulse pressure in those patients cooled to a temperature not less than 28° C. Below this point there was a progressive precipitous drop through the ranges observed, 28° to 26° C. (Fig. 2). On rewarming, the pulse pressure increased above normal for the group as a whole.

Five of the patients showed an acceleration in heart rate immediately postocclusion. The heart rates from this point throughout the occlusion were conditioned by two factors, other than the level of cooling: (1) the coronary perfusion of neostigmine, which slowed the rate; and (2) manipulation of the heart, which created bursts of acceleration, especially idioventricular beats. The heart rate slowed during circulatory occlusion in most patients, the major change usually occurring rather abruptly between ½ and 2 minutes following onset of occlusion, or 15 seconds to 1½ minutes after the perfusion of neostigmine. These data are tabulated in Table V. In general, slightly larger doses were used in patients with the large hearts of atrial septal defects.

TABLE V. CHANGE IN HEART RATE DURING CIRCULATORY OCCLUSION AND DOSAGE OF NEOSTIGMINE USED

| PATIENT | DIAGNOSIS | DOSE OF NEOSTIGMINE (MG.) | HEART RATE | |
|---|---|---|---|---|
| | | | PRIOR TO NEOSTIGMINE | AFTER NEOSTIGMINE |
| P. S. | IASD | 0.25 | 84 | 40 |
| T. G. | IASD | 0.25 | 90 | 42 |
| F. H. | IASD | 0.22 | 50 | 24 |
| H. B. | IASD | 0.20 | 70 | 55 |
| B. J. | IASD<br>IVSD | 0.13 | 80 | 50 |
| J. St. J. | IASD | 0.25 | 80 | 36 |
| E. C. | TF | 0.20 | 80 | 30 |
| S. P. | PVS | 0.22 | 72 | 42 |
| H. T. | PVS | 0.17 | 90 | 60 |
| R. M. | PVS<br>PDA | 0.13 | 60 | 60 |

The venous pressure rose on occlusion in 7 of 8 patients studied (Table VI). In the eighth patient (E. H.), there was no significant rise. As the occlusion was maintained, in 5, the pressure began to fall, in 2 instances (F. H. and J. St. J.), dropping below the preocclusion control levels. In 2 of the subjects (L. H. and H. B.), following the drop there developed a second elevation, occurring during the latter phases of the occlusion.

*Release of Occlusion.*—Upon release of the occlusion in the temperature range 28° to 31° C., 6 patients showed an immediate overshoot of the blood pressure beyond preocclusion control levels. The overshoot developed within 5 to 30 seconds (Table IV). None of the patients below 28° C. developed an immediate overshoot; one patient (H. B.), however, developed a delayed elevation in pressure one minute after release. (One minute may be too long to be classed as a reflex reaction, even at a low heart rate.) After 30 minutes, the blood pressure of all patients (with one exception, R. M. [2]) was as high or higher than preocclusion. Of the 6 patients showing the overshoot, 2 had an accompanying fall in pulse rate, reaching its maximum within one minute of the release. In 3, the pulse rates were stable. Over a 15- to 30-minute period, the heart rates gradually returned to preocclusion [illegible]

TABLE III. VENOUS PRESSURE CHANGES DURING HYPOTHERMIA AND REWARMING

| PATIENT | NORMOTHERMIA |  | HYPOTHERMIA |  |  | REWARM |  |  |
|---|---|---|---|---|---|---|---|---|
|  | TEMPERATURE | V. P. (MM. HG) | TEMPERATURE | V. P. (MM. HG) | DIFFERENCE | TEMPERATURE | V. P. (MM. HG) | DIFFERENCE |
| E. A. | 37 | 3 | 26 | 8 | +5 | 32 | 6 | +3 |
| P. S. | 37 | 3 | 26 | 11 | +8 | - | - | - |
| F. H. | - | - | 26 | 7 | - | 35 | 8 | - |
| H. H. | 37 | 12 | 26 | 4 | -8 | 32 | 4 | -8 |
| R. B. | 37 | 1 | 29 | 5 | +4 | 33 | 3 | +2 |
| S. P. | 37 | 5 | 29 | 7 | +2 | - | - | - |
| J. St. J. | 37 | 7 | 29 | 10 | +3 | 33 | 7 | 0 |
| H. B. | 37 | 8 | 27 | 10 | +2 | 33 | 14 | +6 |

All pressures were taken with the chest intact; the comparisons are with the precooling normal. On cooling, an increase in venous pressure occurs which tends to persist to the late phase of rewarming.

TABLE IV. MEAN ARTERIAL BLOOD PRESSURE AND HEART RATE DURING TOTAL CIRCULATORY OCCLUSION AND RELEASE

| PATIENT DEFECT | TEMPERATURE (°C.) | IMMEDIATE PRE-OCCLUSION |  | OCCLUSION (TIME IN MINUTES) |  |  |  |  |  |  |  |  | RELEASE (TIME IN MINUTES) |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  |  | IMMEDIATE | .25 | .5 | 1 | 2 | 3 | 4 | 5 | 6 | IMMEDIATE | .25 | .5 | 1 | 5 | 15 | 3 |
| . H. | 26 | B.P. | 64 | -- | 55 | -- | 70 | 55 | 47 | 42 | 50 | 42 | 45 | 45 | 56 | 54 | 80 | 76 | 6 |
| IASD |  | H.R. | 60 | -- | 75 | -- | 75 | 30 | 45 | 30 | 30 | 30 | -- | 30 | 30 | 30 | 36 | 36 | 4 |
| . B. | 27 | B. P. | 85 | -- | 36 | 29 | 31 | 25 | 25 | 25 | 29 | 29 | 17 | 61 | 67 | 105 | 89 | 63 | 7 |
| IASD |  | H.R. | 60 | -- | 96 | 90 | 90 | 42 | 20 | 24 | 30 | 42 | 60 | 48 | 36 | 50 | 66 | 66 | 7 |
| . S. | 26 | B.P. | 78 | 59 | 48 | 34 | -- | -- | -- | -- | -- | -- | -- | -- | 20 | 30 | -- | 57 | 7 |
| IASD |  | H.R. | 84 | 84 | 84 | 84 | -- | 40 | 24 | -- | -- | -- | 24 | 30 | 30 | 30 | -- | 36 | 7 |
| . G. | 26 | B.P. | 43 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 18 | 23 | 40 | 34 | 43 | 56 | 6 |
| IASD |  | H.R. | 60 | -- | 50 | 20 | -- | 24 | 24 | 24 | 24 | -- | 24 | 24 | 24 | 24 | 30 | 24 | 3 |
| . H. | 27 | B.P. | 91 | -- | -- | -- | 47 | 29 | 32 | -- | 25 | -- | -- | -- | 87 | 96 | 85 | 46 | 10 |
| LASD |  | H.R. | 120 | -- | -- | -- | -- | 110 | 40 | -- | 50 | -- | -- | -- | 120 | 110 | 110 | -- | 10 |
| . H. | 27 | B.P. | 64 | 61 | -- | 35 | 19 | 24 | 24 | 26 | 19 | -- | -- | -- | 35 | 36 | -- | -- | 6 |
| IPS |  | H.R. | 110 | 80 | 120 | 120 | 110 | 80 | 60 | 60 | 60 | -- | -- | -- | 70 | 40 | -- | -- | 12 |
| . Z. | 27 | B.P. | 95 | -- | -- | -- | 25 | 25 | -- | -- | -- | -- | 56 | -- | 60 | 80 | 68 | 60 | 5 |
| PVS |  | H.R. |  | (Not obtained) |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| J. | 28 | B.P. | 82 | -- | 60 | 54 | 49 | 46 | 40 | 40 | 40 | 40 | 46 | 93 | 69 | 97 | 57* | 80 | 8 |
| IASD |  | H.R. | 80 | -- | 80 | 80 | 70 | 50 | 50 | 30 | 57 | 57 | 40 | 40 | 40 | 40 | 48 | 66 | 7 |
| . M. (2) | 29 | B.P. | 57 | 41 | 49 | 24 | 24 | 24 | 18 | -- | -- | -- | 28 | 47 | 46 | -- | 66 | -- | 5 |
| PVS-PDA |  | H.R. | 72 | 72 | 60 | 60 | -- | 60 | 60 | -- | -- | -- | 64 | -- | -- | -- | 70 | 66 | 7 |
| . P. | 28 | B.P. | 60 | -- | 33 | 25 | 20 | 18 | 18 | 25 | -- | -- | 30 | 28 | 32 | -- | 50 | -- | 8 |
| PVS |  | H.R. | 60 | -- | 66 | 72 | 42 | 42 | 42 | 42 | -- | -- | 66 | 66 | 66 | -- | 54 | -- | 5 |
| . B. (1) | 28 | B.P. | 76 | 79 | 50 | 34 | 28 | 14 | 14 | -- | -- | -- | 90 | -- | -- | -- | -- | -- | 8 |
| TF |  | H.R. | 60 | -- | -- | -- | -- | -- | -- | -- | -- | -- | 72 | -- | -- | -- | -- | -- | 9 |
| . B. | 31 | B.P. | 80 | 48 | 29 | 27 | 27 | 27 | 27 | -- | -- | -- | 60 | 94 | 100 | 100 | 120 | 95 | 8 |
| PVS |  | H.R. | 120 | 100 | 120 | 120 | 48 | 60 | 96 | -- | -- | -- | 130 | 126 | 100 | 100 | 120 | 72 | 7 |
| . C. | 28 | B.P. | 40 | 36 | -- | 27 | 27 | 24 | 19 | 19 | 16 | -- | 27 | 52 | 73 | 71 | -- | -- | 67 |
| TF |  | H.R. | 80 | 50 | -- | 40 | 40 | 80 | 30 | 24 | 28 | -- | 50 | 60 | 50 | 50 | 54 | -- | 6 |
| . St. J. | 28 | B.P. | 69 | 47 | 40 | 27 | 38 | 30 | 30 | 30 | 16 | 21 | 34 | 78 | 116 | 107 | 80 | 73 | 67 |
| IASD |  | H.R. | 54 | 70 | 90 | 80 | 72 | 36 | 30 | 54 | 72 | 54 | 60 | 66 | 80 | 30 | 60 | 60 | 42 |
| I. T. | 29 | B.P. | 59 | -- | 51 | 35 | 30 | 35 | -- | -- | -- | -- | -- | 51 | 53 | 47 | 64 | 85 | 69 |
| PVS |  | H.R. | 72 | -- | 80 | 90 | 60 | 60 | -- | -- | -- | -- | -- | 60 | 60 | 50 | 60 | 72 | 72 |
| . M. (1) | 30 | B.P. | 59 | 24 | 25 | 24 | 26 | 26 | 22 | 20 | -- | -- | 88 | 96 | 103 | -- | 102 | 81 | 78 |
| IASD |  | H.R. |  | (Not obtained) |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |

*Ventricular fibrillation—defibrillated.

B.P.=blood pressure.

H.R.=heart rate.

At the onset of total circulatory occlusion, the blood pressure dropped sharply, and, as occlusion was maintained beyond one minute, dropped still further, although it rarely fell below a mean of 20 mm. Hg. Upon release of occlusion, an immediate overshoot above preocclusion levels was observed in 6 of 9 patients not cooled below 28° C. In the group cooled below 28° C., one of 7 showed an overshoot, and this was a delayed one.

Following onset of occlusion, the heart rate fell in all patients, usually reaching its maximum decrease in from 30 seconds to 2 minutes. Neostigmine was administered by coronary perfusion from 15 seconds to one minute following occlusion. On return of circulation, the pulse rate returned to preocclusion levels after 5 to 15 minutes.

The venous pressure rose on occlusion in 7 of 8 patients, then tended to fall as the occlusion was maintained. Upon release, the pressure tended to return toward preocclusion control levels within 15 to 30 minutes.

## DISCUSSION

Talbott,[13] in his studies on patients cooled in air, noted an initial rise in both blood pressure and pulse rate. Similar observations were made by Collins and Granatelli,[3] by Kossmann,[6] and by Wayburn.[15] It would be difficult to make comparisons between the observations of this report and those just mentioned, due to the fact that our patients were the only ones cooled rapidly by ice water immersion. We observed a transient rise in blood pressure, but no rise in pulse rate—in fact, a fall. The elevation of blood pressure during immersion cooling, with a fall in rate, is a response, we believe, to the intense vasoconstriction which occurs early in the cooling process. The temperature levels at which these elevations were noted varied from 37° to 34° C., sigmoid temperature. At this point, however, the skin vessels are widely dilated due to the direct paralysis of cold. (The skin of the patient is bright pink). The blood pressure thus remains temporarily elevated, even though the skin vessels have become dilated. This maintenance of blood pressure above normal was probably due to persistent vasoconstriction of the internal vessels. The later drop in blood pressure as cooling continues, we believe, was due to a fall in cardiac output and not to a release of the peripheral resistance.

The "clinical disappearance" of the blood pressure and pulse is an interesting phenomenon. It was observed by Collins,[3] Kossmann,[6] and Talbott.[13] Talbott further noted that the blood pressure must have been adequate at the times of "disappearance" since puncture of an artery resulted in a forceful jet of blood. This observation is confirmed in the present study. The blood pressure was lost first and then the pulse. The mechanism of the "clinical disappearance," we believe, lies in the physical state of the large arteries during cooling. Early in cooling the larger vessels are unaffected because the blood temperature has not appreciably changed. Not until the intense peripheral vasodilatation has occurred does the blood begin to cool. Circulation of cool blood through human arteries causes intense constriction of these vessels, as has been demonstrated by Bazett and his associates.[1] Thus, once the

The periods of occlusion (between 2 and 6 minutes) bore no direct relationship to the development, degree, or recovery of the blood pressure overshoot. Neither the basal preocclusion pressure levels nor the type of cardiac malformation appeared to have any effect on the presence or character of the overshoot.

The venous pressures fell immediately upon release of occlusion (Table VI), and by 30 minutes were at or below preocclusion levels, except R. M. (1) in whom the pressure remained elevated.

TABLE VI. VENOUS PRESSURE (MM. HG) DURING OCCLUSION AND ON RELEASE (OPEN CHEST)

| PATIENT | TEMPERATURE °C. | PREOCCLUSION HYPOTHERMIA | OCCLUSION (TIME IN MINUTES) | | | | | | | | RELEASE (TIME IN MINUTES) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | IMMEDIATE | .5 | 1 | 2 | 3 | 4 | 5 | 6 | IMMEDIATE | .5 | 1 | 5 | 15 | 30 |
| P. S.<br>IASD | 26 | 13 | 19 | 18 | 18 | 18 | 16 | -- | -- | -- | 13 | 8 | 8 | -- | 4 | 5 |
| F. H.<br>IASD | 26 | 9 | 10 | 10 | 10 | 6 | 3 | 2 | 3 | 4 | 5 | 9 | 10 | 10 | 10 | 7 |
| H. B.<br>IASD | 27 | 8 | 22 | 22 | 19 | 20 | 21 | 21 | 27 | 27 | 15 | 10 | 10 | 8 | 10 | 10 |
| C. Z.<br>PVS | 27 | 14 | 29 | -- | 21 | 22 | -- | -- | -- | -- | 9 | -- | 10 | 10 | 9 | -- |
| C. R.<br>IASD | 27 | 3 | -- | -- | 23 | 19 | 21 | -- | -- | -- | 8 | -- | 8 | 7 | -- | 0 |
| L. H.<br>IPS | 27 | 2 | -- | -- | -- | 8 | 5 | 5 | 9 | -- | -- | 8 | 8 | -- | -- | 3 |
| J. St. J.<br>IASD | 28 | 10 | 10 | 21 | 21 | 14 | 10 | 4 | 1 | 1 | 1 | 0 | -1 | 0 | 0 | 0 |
| R. M. (1)<br>IASD | 30 | 5 | 28 | 21 | 21 | 23 | 21 | 19 | -- | -- | -- | -- | 19 | 16 | 8 | 12 |

## SUMMARY OF RESULTS

The blood pressure and pulse could not be obtained by the usual clinical methods after various levels of cooling had been achieved. Direct arterial tracings, however, demonstrated adequate blood pressures and rates at the times of "clinical disappearance." On rewarming, the blood pressure and pulse "reappeared" usually at a lower temperature than that at which each had previously been lost to auscultation or palpation. During cooling, an early transient elevation in blood pressure was observed while the heart rate declined progressively. The blood pressure subsequently began to fall, and stabilized at its lowest level 30 minutes after the sigmoid temperature had become stabilized. The heart rate, however, reached its lowest level at the same time as the temperature. The magnitude of blood pressure and pulse rate changes varied considerably from patient to patient in all temperature ranges. The pulse pressures declined in the lower temperature ranges (below 28° C.). Rewarming with diathermy was discontinued at 34° C., at which time, however, the blood pressure and pulse rate were substantially within normal limits for the group as a whole.

The venous pressure became elevated during hypothermia. On rewarming, a continued elevation of the venous pressure above normal occasionally remained.

blood in an already overloaded constricted system. It is of interest that in 2 of the 4 cases, as the occlusion was maintained, the venous pressure fell below the preocclusion level and, in one of these, even below the normothermic level. The explanation for the venous pressure fall while the occlusion was maintained is conjectural.

Upon release of occlusion and return of circulation, the blood pressure showed an interesting immediate elevation above preocclusion levels in 5 patients, a phenomenon we have referred to as "overshoot." In 2 patients, the peak of the overshoot coincided with a slowing of pulse rate. This reaction is not unlike the course of events following the Valsalva maneuver in normal individuals.[5, 8] The principal post-Valsalva circulatory events are mainly reflex in character, as are those following release from total circulatory occlusion. The differences noted are primarily quantitative and are possibly related to the body temperature level; i.e., in hypothermia the magnitude of response is less and the time interval of the events prolonged. For example, during normothermia following release of a Valsalva maneuver, the overshoot reaches its peak in 4 or 5 seconds at a normal heart rate of 70 to 90 per minute.[8] During hypothermia, the time required to reach the peak varied between 5 and 30 seconds, with the majority requiring over 15 seconds, but with heart rates of only 30 to 50. No such overshoot was seen in 3 patients whose temperatures fell to 26° C. It is possible that this degree of hypothermia is sufficient to inhibit completely the reflex pathways involved in the response, although some baroceptor responses appear to be still present, at this temperature level.[10]

By and large, however, it may be concluded on the basis of the data presented that the cardiovascular reflex mechanisms regulating the circulation are intact in the range of hypothermia studied (26 to 31° C.), although they may be somewhat slowed.

## SUMMARY AND CONCLUSIONS

1. The effects on the cardiovascular system of cooling and rewarming were studied in 23 patients undergoing hypothermia, total circulatory occlusion, and intracardiac surgery.
2. Upon cooling there was a transient elevation in blood pressure, followed by a fall which was not maximum until 30 minutes after sigmoid temperature had reached its lowest point.
3. The pulse rate fell progressively with cooling and the temperature and rate reached lowest levels simultaneously.
4. Blood pressures and pulses were lost to auscultation and palpation at varying points during cooling. At the times of the "clinical disappearance," both blood pressure and pulse rate were quite adequate, as demonstrated by direct measurements. The mechanism of this phenomenon is discussed.
5. The magnitude of eventual blood pressure and heart rate changes varied considerably between patients and appeared to have no direct relationship to age and type of heart disease.

blood begins to cool, the larger arteries become rigid, and the mechanism essential to auscultatory blood pressure determinations[16] is lost. The phenomenon has nothing to do with pulse pressure, which does not show any significant change until long after the blood pressure "disappearance" has occurred. A pulse wave, of course, persists at least for a time, and is palpable. Thus, the palpable pulse is lost after the blood pressure.

It is important to realize that even though these clinical signs "disappear," the blood pressure and pulse are quite adequate as measured directly. This emphasizes the value, in fact the necessity, of monitoring heart rate by means of electrocardiography through the cooling experience. In addition, the presence of an inlying arterial cannula and pressure recording device is highly desirable.

The observed elevation in venous pressure which begins during cooling and persists throughout the experience might be explained as follows. As the cardiac output progressively falls during cooling, blood accumulates on the venous side of the systemic circulation. Normally, this would cause little change in pressure until severe venous distention occurred because of expansion in venous reservoirs. These changes in venomotor tone are normal reflex responses to volume changes.[16] As the blood cools, however, the same constrictive effect seen in the arteries occurs in the veins.[1] Under these circumstances, the flexibility of the venous reservoirs is lost and volume changes are reflected in rising pressure. Venous pressure still remains high, however, even after stabilization of cardiac output when hypothermia has been achieved and also during rewarming when cardiac output is actually increasing. The explanation for this is not at all clear.

During total circulatory occlusion, the blood pressure reached its low point within 2 minutes, yet in most instances remained well above zero, despite the fact that there was absolutely no cardiac output. Whether osmotic phenomena play a role in this "cadaver" pressure, we cannot say.

It was noted that the heart rate fell acutely, usually after 30 seconds to 2 minutes of occlusion. On the basis of previously reported experimental evidence,[12] it is our uniform practice to cross-clamp the aorta about one inch from the heart and inject about 0.2 mg. of neostigmine into the root of the aorta to perfuse the coronary circuit with the drug. This maneuver is usually completed at 45 seconds to one minute of occlusion. We believe this agent was an important factor in the observed slowing of the heart, although inflow occlusion alone may also play a part. That it is possible to slow or, in fact, stop the heart with neostigmine is well known.[14] In the observations reported here, the decrease in heart rate occurred usually within 15 or 90 seconds following completion of the neostigmine injection. This agent may also have been a factor in the slow return of rate following release of occlusion in some patients.

The venous pressure prior to occlusion was elevated above normal. Upon total circulatory occlusion, a further rise in pressure of considerable magnitude was usually seen. This would appear to be due to sudden damming of

# ARTERIOVENOUS FISTULA OF THE RIGHT INTERNAL MAMMARY VESSELS FOLLOWING RADICAL MASTECTOMY VISUALIZATION BY ANGIOCARDIOGRAPHY

FRANK GLENN, M D, AND ISRAEL STEINBERG, M D
NEW YORK, N Y

TRAUMA incident to en masse ligation of vessels during surgical operations has become recognized as a cause of arteriovenous fistulas In the case herein described an internal mammary arteriovenous fistula developed following right radical mastectomy for carcinoma The arteriovenous fistula was visualized by the method of angiocardiography At operation, a silk ligature was found around the vessels, individual ligation of the vessels and excision of the fistulous sac were curative To our knowledge, an arteriovenous fistula of the internal mammary vessels following surgery has not been previously reported

## CASE REPORT

A 48 year old housewife (N Y H No 74274) was admitted on Sept 20, 1955, complaining of a continuous vibration of the right anterior chest and a buzzing noise along the right side of the neck Two years prior to entry she had had a right radical mastectomy at another hospital because of cancer of the breast The operative note indicated that the procedure was carried out in a routine manner and without difficulty, there was no excessive bleeding The postoperative course was uneventful except for pain in the anterior chest which persisted for several months Six months following operation tenderness and vibrations over the right anterior chest and a whistling noise along the right neck began Despite these symptoms, several months elapsed before the characteristic bruit was recognized and the diagnosis of arteriovenous fistula made

In the past, the patient had been well except for dysmenorrhea The only significant illness was the carcinoma of the breast at age of 46 years Physical examination revealed a well developed and well nourished woman in no distress Examination of the thorax disclosed a well healed scar of a right radical mastectomy, the pectoral muscles were absent In the second right anterior interspace near the sternum, there was a 5 by 5 cm swelling which felt soft and contained a coarse thrill A loud bruit was clearly audible, loud, rough machinery like systolic and diastolic murmurs were heard with the stethoscope The heart was normal in size and there were no cardiac murmurs The blood pressure was elevated, 194/104 mm Hg The lungs were clear A few firm, small axillary and inguinal lymph nodes were palpable The rest of the examination, including the pelvic organs, was normal

Laboratory data, including x ray examinations of the heart, lungs, sternum, ribs, thoracic spine, and long bones, were normal Angiocardiography was done in the frontal and lateral positions on Sept 20, 1955 Films were made at one second intervals and showed the right innominate vein, superior vena cava, right atrium and ventricle, pulmonary arteries and branches to be normal The pulmonary veins, left atrium and ventricle,

---

From the Departments of Surgery and Radiology The New York Hospital Cornell Medical Center

Received for publication June 18 1956

6. Venous pressure rose on cooling and remained elevated. Possible mechanisms underlying this phenomenon are discussed.

7. During total circulatory occlusion, the blood pressure fell, the heart rate (following neostigmine injection) slowed abruptly, and venous pressure at first rose, then fell in some instances.

8. Following return of circulation, an immediate overshoot of blood pressure occurred, while venous pressure dropped abruptly.

9. During rewarming, all the circulatory signs have returned to within normal limits at 34° C.

10. Cardiovascular reflexes governing the circulation appear to be intact and adequate in human beings undergoing hypothermia in the range 26° to 31° C.

## REFERENCES

1. Bazett, H. C., Love, L., Newton, M., Day, R., and Forster, R. E.: Temperature Changes in Blood Flowing in Arteries and Veins in Man, J. Appl. Physiol. 1: 3-19, 1948.
2. Bigelow, W. G., Mustard, W. J., and Evans, J. G.: Some Physiologic Concepts of Hypothermia and Their Application to Cardiac Surgery, J. THORACIC SURG. 28: 463-472, 1954.
3. Collins, V. J., and Granatelli, A.: Controlled Hypothermia During Anesthesia in Human Adults, Angiology 6: 118-128, 1955.
4. Cookson, B. A., Neptune, W. B., and Bailey, C. P.: Intra-Cardiac Surgery and Hypothermia, J. Internat. Coll. Surgeons 18: 685-694, 1952.
5. Elisberg, E. I., Miller, G., Weinberg, S. L., and Katz, L. N.: The Effect of the Valsalva Maneuver on the Circulation. II. The Role of the Autonomic Nervous System in the Production of the Overshoot, Am. Heart J. 45: 227-236, 1953.
6. Kossmann, C. E.: General Crymotherapy: Cardiovascular Effects, Bull. New York Acad. Med. 16: 317-340, 1940.
7. Lewis, F. J.: Hypothermia in Cardiac and General Surgery, Minnesota Med. 38: 77-81, 1955.
8. McIntosh, H. D., Burnum, J. F., Hickam, J. B., and Warren, J. V.: Circulatory Changes Produced by the Valsalva Maneuver in Normal Subjects, Patients With Mitral Stenosis and Autonomic Nervous System Alterations, Circulation 9: 511-520, 1954.
9. Montgomery, A. V., Prevedel, A. E., and Swan, H.: Prostigmine Inhibition of Ventricular Fibrillation in the Hypothermic Dog, Circulation 10: 721-727, 1954.
10. Nashat, F. S., and Neil, E.: The Effect of Hypothermia on Baroceptor and Chemoceptor Reflexes, J. Physiol. 127: 59-60, 1955.
11. Smith, L. W., and Fay, T.: Observations on Human Beings With Cancer Maintained at Reduced Temperatures of 75-90° F., Am. J. Clin. Path. 10: 1-11, 1940.
12. Swan, H., Virtue, R. W., Blount, S. G., Jr., and Kircher, L. T.: Hypothermia in Surgery. Analysis of 100 Clinical Cases, Ann. Surg. 142: 382-400, 1955.
13. Talbott, J. H.: The Physiologic and Therapeutic Effects of Hypothermia, New England J. Med. 224: 281-288, 1941.
14. U.S. Pharmacopeia: Effects of Prostigmine, XV: 455-457, 1955.
15. Wayburn, E.: Immersion Hypothermia, Arch. Int. Med. 70: 77-91, 1947.
16. Wiggers, C. J.: Physiology in Health and Disease, ed. 5, Philadelphia, 1951, Lea & Febiger, pp. 627-631.

and aorta were also of normal size and contour. At the time of filling of the aorta and brachiocephalic arteries, the right internal mammary artery was also visualized and its termination in a cluster of vessels measuring about 1.5 cm. in diameter was clearly demonstrated (Fig. 1).

At operation on Sept. 23, 1955, drapings were arranged to expose the entire anterior chest wall from the level of the clavicles down to the costal margins and between the anterior axillary lines. At the level of the third right interspace there was a 5 by 5 cm. pulsatile area raised about ½ cm. above the tissues. On palpation, a thrill was felt, and compression against the third rib caused it to disappear.

A skin flap with its base cephalad and just large enough to incorporate the lesion was made. The skin flap was dissected free and a pulsating mass exposed. The third rib was then resected from its junction with the sternum for a distance of about 12 cm. The right internal mammary vessels were visualized and the arteriovenous fistula inspected. When the fistula was dissected apart, a black suture, probably the site of ligature and cause of the arteriovenous fistula was revealed (Fig. 2). The arteriovenous fistula was dissected free and the individual vessels ligated. Complete collapse of the fistulous sac resulted; there was also disappearance of the thrill and murmurs. Numerous perforating branches from the intercostal vessels were individually ligated allowing removal of the fistula and aneurysmal sac. The skin flap was then brought into position and secured with interrupted sutures of pure silk to the subcutaneous tissue and skin. A pressure dressing with rubber sponge was applied to avoid accumulation of blood and serum under the skin flaps. Recovery was uneventful and the patient was discharged on the twelfth postoperative day.

Pathologic study of the specimen revealed a saclike structure measuring 2 by 1.8 by 1.5 cm.; several "feeder" vessels were present. Microscopic study showed the sac to be composed of a thick layer of connective tissue with connecting endothelium lined blood vessels.

### COMMENT

Injuries to blood vessels have been recorded ever since the beginning of medical history and go back to the era of blood letting. Elkin[1] aptly remarked that although blood letting has long passed into oblivion, a new evil, the large scale use of venous and arterial punctures for diagnosis and treatment may be expected to be followed by an increased number of aneurysms and fistulas. However, such complications have been rare. Trauma to blood vessels during surgical procedures is common; transfixion and ligation of individual vessels are standard procedures. Ligation of arteries and veins en masse, however, is particularly liable to give rise to fistulas. Holman[2] also has emphasized the danger of erosion of ligatures, particularly after mild infection. It is generally agreed that traction pins, wires, and fixation devices are dangerous in the vicinity of blood vessels because of their tendency to erode vessels and cause arteriovenous fistulas.[3, 4]

Arteriovenous fistulas have been described following thoracentesis,[5] amputations,[6] hysterectomy,[7] thyroidectomy,[8] laminectomy for intervertebral discs,[9] and nephrectomy.[10] Elkin[1] reported three instances of aneurysm and 3 cases of arteriovenous fistulas following operations or surgical procedures in 650 personally observed cases, a total incidence of one per cent.

In the case herein reported, the onset with pulsation in the chest wall and vascular bruit following mastectomy was characteristic of the development of a traumatic, postsurgery arteriovenous fistula. Localization of the lesion to

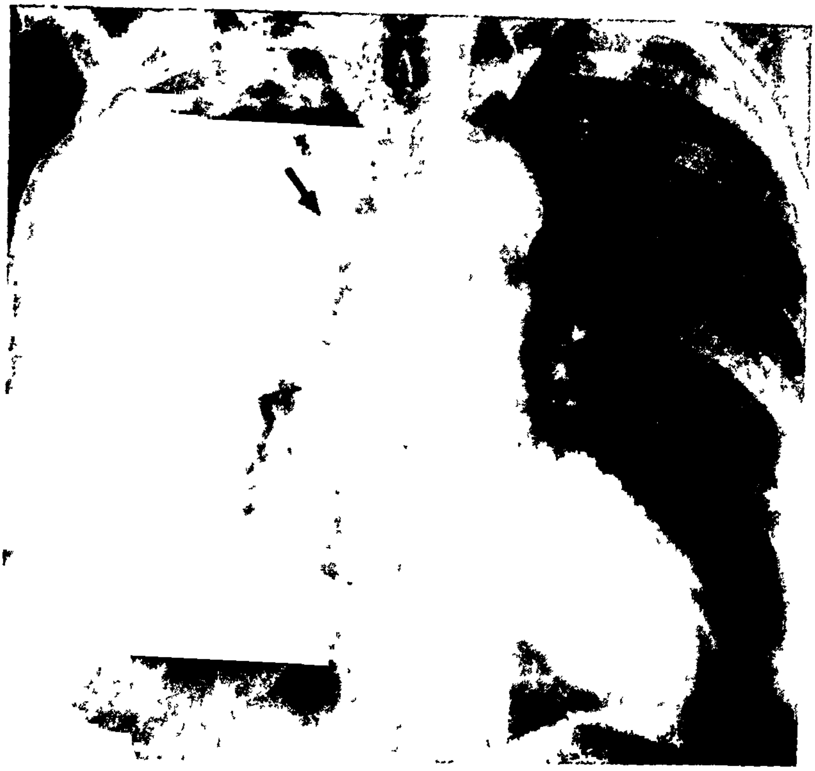


Fig. 1.—Angiocardiogram (frontal view) made 9 seconds a[illegible] of injection shows opacification of the left ventricle and aorta. The [illegible] including the right internal mammary artery (arrow) are clearly [illegible] cluster of opacified vessels at termination of the right internal mammary artery, the site of the arteriovenous fistula

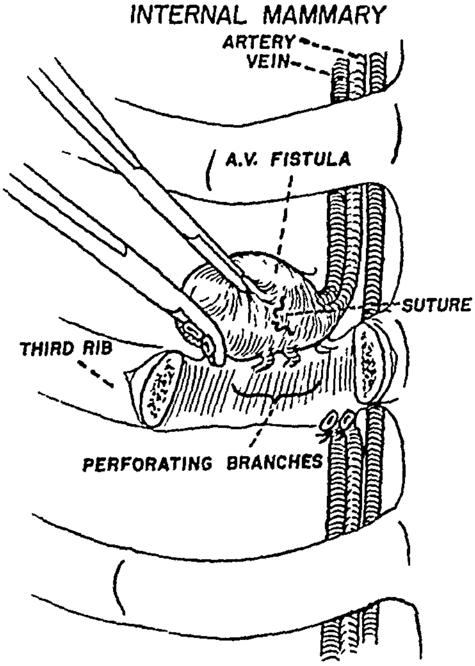


Fig. 2.—Sketch of the operative findings of the right internal mammary arteriovenous fistula which developed after radical mastectomy.

# PERICARDIAL FAT NECROSIS

## Report of Three Cases

Robert C. Jackson, M.D.,* O. Theron Clagett, M.D.,** and
John R. McDonald, M.D.***
Rochester, Minn.

EXPLORATORY thoracotomy has justifiably become an increasingly common and accepted procedure. It is frequently the most expedient or the only method available to establish a definitive diagnosis, without which intelligent management is impossible. The following three clinically indeterminate chest lesions were surgically excised and are reported because of their unusual nature. They represent a clinicopathologic entity to which we can find no reference in the medical literature.

### REPORT OF CASES

CASE 1.—A 52-year-old white male farmer registered at the Mayo Clinic on Aug. 23, 1951. For approximately one year he had experienced intermittent episodes of severe sharp lancinating pain in the lower aspect of his right anterior chest. In June, 1951, he had been hospitalized for a particularly severe attack of constant pain which was aggravated by deep breathing. A thoracic roentgenogram was interpreted as normal at that time, but a repeat study in August revealed a lesion in the right hemithorax and he was referred to us for evaluation. He gave a history of pneumonia with pleurisy on the right side in 1929 and removal of a squamous cell epithelioma from the left eyelid in May, 1951. A mobile unit roentgenogram of the thorax in 1949 had been normal.

On physical examination the patient appeared in excellent general health, except for moderate obesity. He was 66 inches in height and weighed 185 pounds. Definite point tenderness to palpation was present over the costochondral junction of the right fifth rib. No other significant findings were noted.

No abnormal data were recorded in a thorough laboratory study including an electrocardiogram. Posteroanterior and left anterior oblique roentgenograms of the chest (Fig. 1) demonstrated a mass over the dome of the right diaphragm in continuity with the chest wall.

On Aug. 29, 1951, a right anterolateral incision was made and the chest was entered through the bed of the fifth rib. A firm mass of lobulated fat was found surrounded by a moderate degree of inflammatory reaction fixing it to the diaphragm, the anterior chest wall, and the right lower lobe. Curiously, it was attached to the pericardium by a pedicle 3 cm. in length and 0.7 cm. in diameter. This was ligated, and the mass was removed after it had been freed by means of sharp dissection. An unusually large amount of lobulated fat was noted overlying the parietal pericardium.

---

The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

Received for publication June 27, 1956.

*Fellow in Thoracic Surgery, Mayo Foundation.

**Section of Surgery, Mayo Clinic and Mayo Foundation.

***Section of Surgical Pathology, Mayo Clinic and Mayo Foundation.

the right internal mammary vessels was easily accomplished by the intravenous method of angiocardiography.[11] Furthermore, visualization of the arteriovenous fistula excluded other (congenital) types such as chest wall,[12] pulmonary,[13] and coronary.[14] Finally, the normal cardiovascular findings also eliminated patent ductus arteriosus, aortic septal defect, and rupture of an aortic sinus aneurysm. The finding of an old ligature in the immediate vicinity of the fistula is strong evidence that erosion was responsible for formation of the arteriovenous connection. Surgical excision of acquired arteriovenous fistulas with ligation of the involved vessels is curative.

## ADDENDUM

Since this paper was submitted for publication, Muller and Goodwin[15] reported operating on a patient with a renal arteriovenous fistula which was discovered 29 years after nephrectomy.

## REFERENCES

1. Elkin, D. C.: Aneurysm Following Surgical Procedures: Report of 5 Cases, Ann. Surg. 127: 769, 1948.
2. Holman, E.: Arteriovenous Aneurysm: Abnormal Communications Between the Arterial and Venous Circulations, New York, 1937, The Macmillan Co.
3. Greeley, P. W., and Throndson, A. H.: Arteriovenous Aneurysm Resulting From Application of a Roger Anderson Splint, J. A. M. A. 124: 1128, 1944.
4. Gamm, K. E.: Arteriovenous Fistula, J. A. M. A. 119: 134, 1942.
5. Reid, M. R., and McGuire, J.: Arteriovenous Aneurysms, Ann. Surg. 108: 643, 1938.
   Elkin, D. C.: Arteriovenous Aneurysm of the Phrenic Vessels: Report of a Case Following Thoracentesis, J. A. M. A. 141: 531, 1949.
6. Stuart, D. W.: Arteriovenous Aneurysm Following Amputation, Brit. M. J. 2: 346, 1929.
   Mason, J. M., Pool, R. M., and Collier, J. P.: The Treatment of Traumatic Arteriovenous Aneurysms, South. M. J. 29: 248, 1936.
7. Elkin, D. C., and Banner, E. A.: Arteriovenous Aneurysm Following Surgical Operations, J. A. M. A. 131: 1117, 1946.
8. Downes, W. A.: Arteriovenous Aneurysm of the Superior Thyroid Artery and Vein, Ann. Surg. 59: 789, 1914.
   Mora, J. M.: Arteriovenous Aneurysm of Left Superior Thyroid Vessels, Surg., Gynec. & Obst. 48: 123, 1929.
   Selman, J. J., and Freedlander, S. O.: Arteriovenous Aneurysm of Thyroid Vessels, Am. J. Surg. 17: 99, 1932.
   Ransohoff, J. L.: Arteriovenous Aneurysm of Superior Thyroid Artery and Vein, Surg., Gynec. & Obst. 61: 816, 1935.
9. Linton, R. R., and White, P. D.: Arteriovenous Fistula Between Right Common Iliac Artery and Inferior Vena Cava: Report of a Case of Its Occurrence Following an Operation for a Ruptured Intervertebral Disc With Cure by Operation, Arch. Surg. 50: 6, 1945.
   Glass, B. A., and Ilgenfritz, H. C.: Arteriovenous Fistula Secondary to Operation for Ruptured Intervertebral Disc, Ann. Surg. 140: 122, 1954.
10. Hollingsworth, E. W.: Arteriovenous Fistula of the Renal Vessels, Am. J. M. Sci. 188: 399, 1934.
11. Robb, G. P., and Steinberg, I.: Visualization of the Chambers of the Heart, the Pulmonary Circulation and the Great Blood Vessels in Man: A Practical Method, Am. J. Roentgenol. 41: 1, 1939.
    Steinberg, M. F., Grishman, A., and Sussman, M. L.: The Angiocardiographic Demonstration of an Arteriovenous Fistula, Surg., Gynec. & Obst. 95: 93, 1942.
12. Maier, H. C., and Stout, A. P.: Congenital Arteriovenous Fistulas of the Thoracic Wall, Circulation 1: 809, 1950.
13. Steinberg, I., and McClenahan, J.: Pulmonary Arteriovenous Fistulas: Angiocardiographic Observations in Nine Cases, Am. J. Med. 19: 549-568, 1955.
14. Davis, C., Dillon, R. F., Fell, E. H., and Gasul, B. M.: Anomalous Coronary Artery Simulating Patent Ductus Arteriosus, J. A. M. A. 160: 1047, 1956.
15. Muller, W. H., Jr., and Goodwin, W. E.: Renal Arteriovenous Fistula Following Nephrectomy, Ann. Surg. 144: 240-244, 1956.

The gross specimen measured 5 by 2.5 by 2 cm. It was firm and was composed entirely of fat, which was medium to light brown. Microscopic sections (Fig. 2, *a*) demonstrated fat necrosis. There were definite phagocytosis of some of the fat and pooling of the lipides in large spaces, frequently 10 to 20 times the diameter of a normal fat cell. A moderate amount of fibrous tissue was present in heavy strands and islands throughout the specimen. There was very little inflammatory exudate. Serial block sections were prepared with elastic van Gieson stain (Fig. 2, *b*). Some of the arterioles demonstrated a severe degree of subintimal fibrosis and others a mild to moderate medial hypertrophy with consequent narrowing of the lumina. Several small venules had thrombosed and partially recanalized. The majority of the vessels, however, were quite normal.

Case 2.—A 63-year old white male truck driver registered on May 2, 1955. Six weeks previously a very severe pain had abruptly developed in the left precordium. He had had a myocardial infarction four years previously, and both he and his physician thought he was suffering from a similar attack. A low-grade fever and nonproductive cough developed during subsequent hospitalization, and an electrocardiogram showed no change. The pain gradually diminished and became intermittent, but was referred to the left shoulder and was aggravated by a cough. Thoracic roentgenograms demonstrated a lesion which did not change over a 6-week interval, despite antibiotic therapy. Consequently, he was referred to the Clinic for further study. His past medical record included a coronary occlusion in 1951 and ligation of the left saphenous vein in 1947. Chest roentgenograms in 1952 and 1953 were normal.

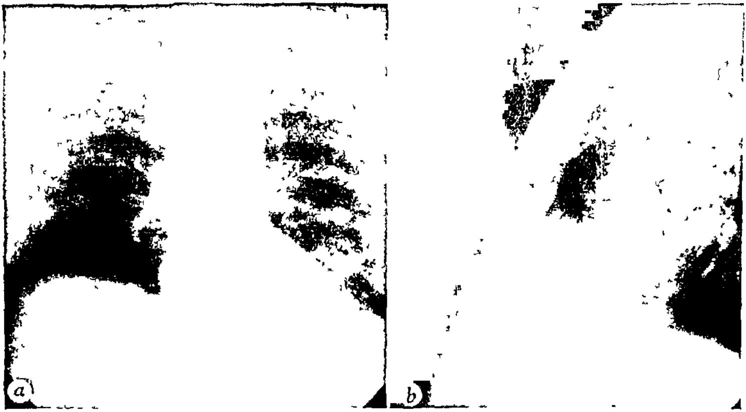


Fig 3—Case 2. Posteroanterior (*a*) and left lateral (*b*) roentgenograms, showing rounded opacity overlying the left heart border and anterior against the chest wall

Physical examination revealed a somewhat obese, mentally depressed man, who did not appear ill. He was 70 inches tall and weighed 220 pounds. Moderate benign prostatic hypertrophy was noted, but other findings were quite normal. His exercise tolerance was excellent.

Laboratory studies were within the usual range, except for an electrocardiographic tracing indicative of an ancient healed posterior myocardial infarct. Posteroanterior and lateral thoracic roentgenograms (Fig. 3) demonstrated a rounded juxtacardiac mass.

On May 6, 1955, a left posterolateral incision was made through the bed of the sixth rib. A firm, ovoid mass was encountered, which was densely adherent to the terior c

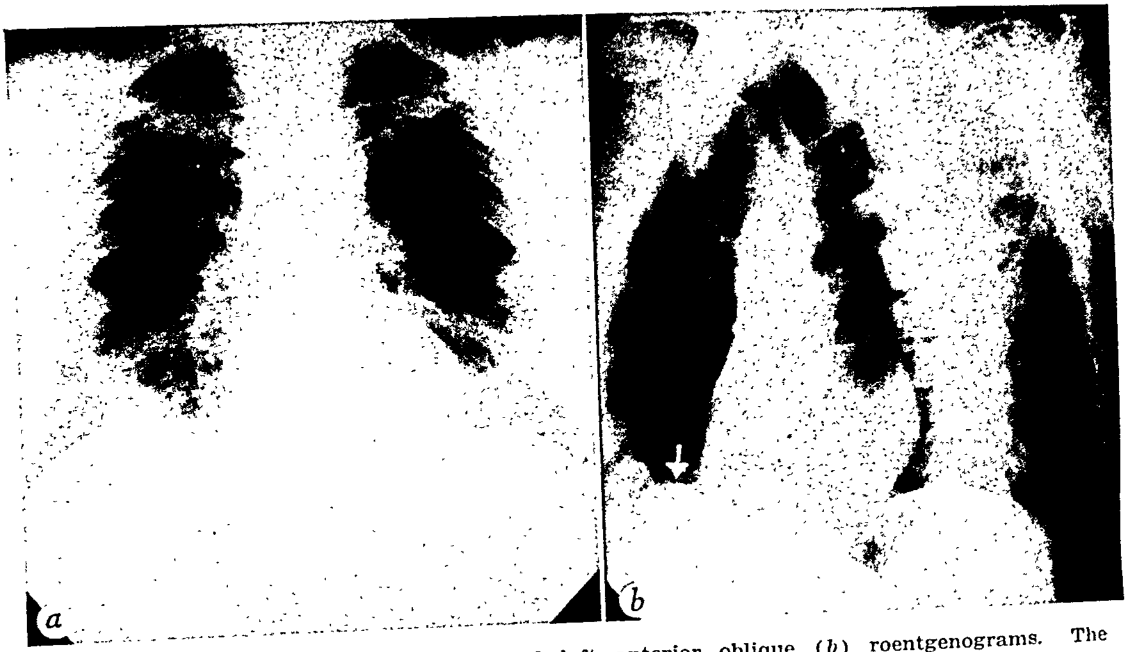


Fig 1.—Case 1. Posteroanterior (*a*) and left anterior oblique (*b*) roentgenograms. The pedicle found at operation is clearly demonstrated in *b* (arrow).

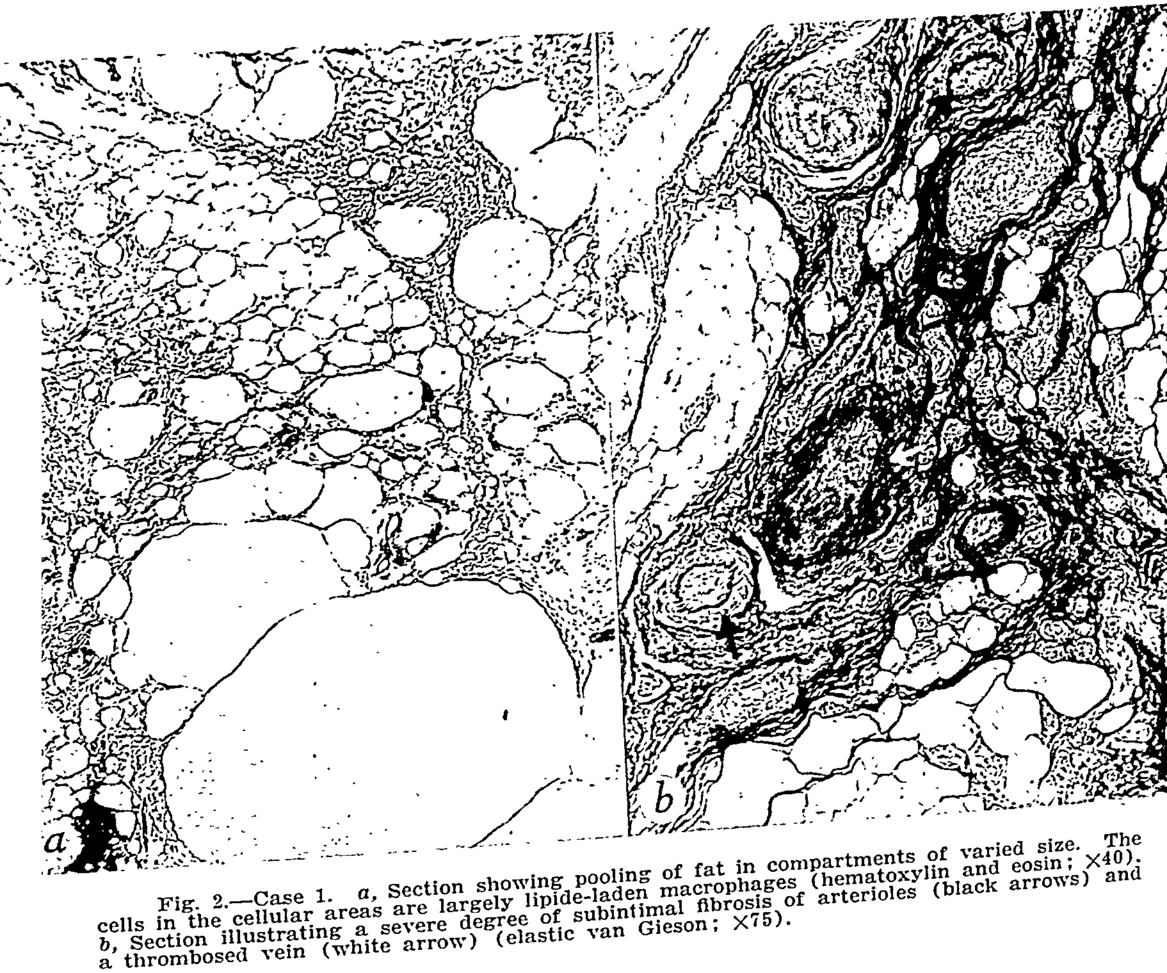


Fig. 2.—Case 1. *a*, Section showing pooling of fat in compartments of varied size. The cells in the cellular areas are largely lipide-laden macrophages (hematoxylin and eosin; ×40). *b*, Section illustrating a severe degree of subintimal fibrosis of arterioles (black arrows) and a thrombosed vein (white arrow) (elastic van Gieson; ×75).

history, primary carcinoma was considered to be a most unlikely possibility. After a frank discussion with both the patient and her husband, an observation period of one month was advised.

Readmission roentgenograms on Sept. 9, 1955, demonstrated no appreciable change in the lesion. Thoracotomy was then recommended and on Sept. 14, 1955, the left lung was exposed through the bed of the sixth rib. No free fluid was encountered, and the pleural space was free except in the region of the diaphragm, where edematous adhesions were encountered. A firm, ovoid mass could be palpated low in the interlobar fissure.

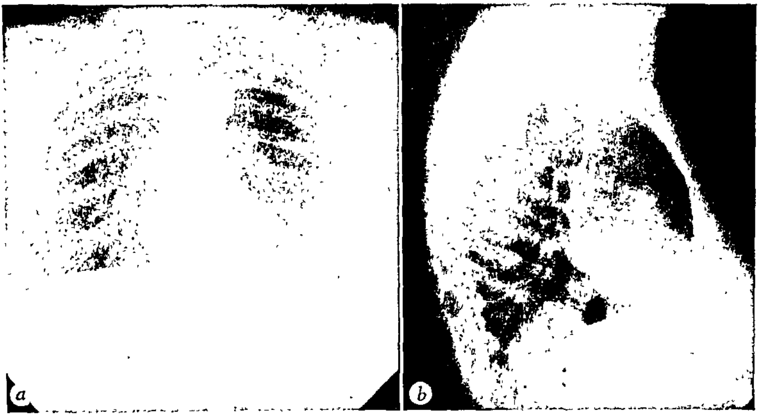


Fig. 5.—Case 3. *a*, Posteroanterior view, showing opacity due to fat necrosis overlying the elevated left hemidiaphragm. *b*, Lateral view, showing lesion in the area of the interlobar fissure and overlain by the base of the heart.

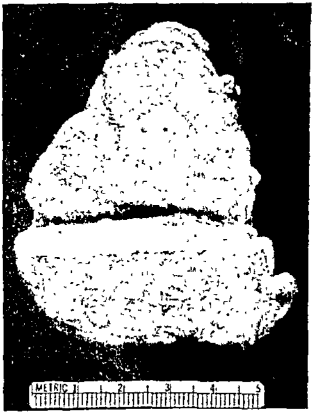


Fig. 6.—Case 3. Surgically resected specimen of pericardial fat. The fat is bulkier and firmer than normal.

wall, pericardium, and lung. It appeared to originate in pericardial fat and was dissected away and removed without damage to the lung or the pericardium.

The surgical specimen (Fig. 4) measured 8 by 4.5 by 2.5 cm. and consisted of pale brown fat, which was abnormally firm. Histologically, the findings were similar to those in Case 1 in that fat necrosis was present. Both the dissolution of fat and the fibrotic process were less extensive in this specimen than in Case 1. The fatty envelopes were filled with phagocytized fat, and a moderate inflammatory reaction was present. Serial block sections prepared with elastic van Gieson stain demonstrated vascular changes similar to those in Case 1, but of lesser degree.

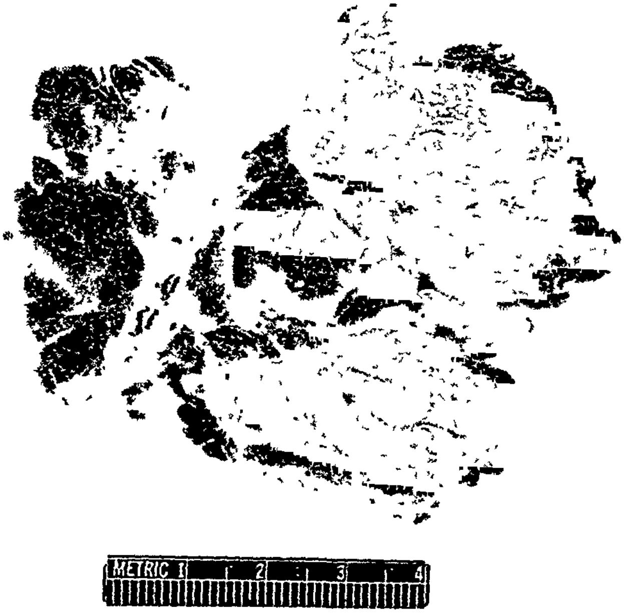


Fig. 4.—Case 2. Gross specimen of resected pericardial fat.

Case 3.—A 47-year-old farmer's wife was referred to us on Aug. 8, 1955. Her health had been excellent until July 14, 1955, at which time she experienced a sudden dull constant pain in her left anterior chest. She was immediately hospitalized with a tentative diagnosis of myocardial infarction. The following day the pain had become more severe, was referred to the left shoulder and seemed somewhat aggravated by deep breathing. Chest roentgenograms taken at this time were interpreted as "pleuritis with effusion," but two attempts at thoracentesis yielded only a scanty amount of bloody fluid. Gradual abatement of pain occurred during nine days of hospitalization, and she had been asymptomatic for several days on admission to the Clinic. Her past medical history was not significant, and she had not had a chest roentgenogram prior to her present illness.

The only remarkable finding on physical examination was an extreme degree of obesity. She weighed 215 pounds and was only 62 inches tall.

Routine laboratory studies, including an electrocardiogram, were within the normal range, except for a leukocyte count of 10,900 per cubic millimeter with a normal differential count. Posteroanterior and lateral roentgenograms of the chest (Fig. 5) demonstrated an elevated left hemidiaphragm with an overlying mass in the region of the interlobar fissure. Motion of the posterior portion of the left hemidiaphragm was demonstrated by fluoroscopy, but its anterior portion and the lesion both appeared fixed.

Localized pleuritis with effusion, or the residual of a pulmonary embolism, seemed to be the most likely diagnosis at that time. In view of the patient's sex and recent acute was eliminated with the aid of serial electrocardiograms and thoracic roentgenograms. Many other disease entities might well be considered in the differential diagnosis of this condition; particularly, pulmonary infarction, localized pleural effusion, mesothelioma, pleuropericardial cyst and, as always, primary or metastatic carcinoma.

A consideration of the possible etiology of this entity appears justified. The tissue changes are not consistent with sepsis, and bacteriologic study of two specimens was unrewarding. The pedicle described in our first case appeared at operation to be slightly twisted. This immediately suggests compromise of the vasculature with resultant ischemia. A similar set of circumstances is well known to occur in the appendices epiploicae and in the omentum, but the end result, as would be anticipated, is hemorrhagic infarction and gangrene. Epiploic appendicitis of lesser magnitude does occur, but torsion has not been described in these cases. The vascular changes present in each of our specimens (see report of cases) were somewhat confusing to us. If this entity were of primary vascular origin, one would anticipate a uniform type of vessel abnormality rather than the array of changes which we observed. In an attempt to further evaluate this aspect of the problem, we studied two groups of cases. Eleven surgical specimens of fat necrosis of the breast and six surgically removed epiploic appendicitis specimens were prepared with elastic van Gieson stain. We were considerably surprised to find vessel changes varying from mild to severe in six of the breast and in four of the epiploic appendage specimens. The changes (Fig. 7) were quite similar to those found in our pericardial fat specimens. Apparently nonspecific vascular changes occur frequently in the presence of fat necrosis, and probably as a secondary phenomenon. We are, therefore, unable to offer a reasonable explanation of why fat necrosis should occur in this unusual location.

## SUMMARY

1. Three surgically treated cases of fat necrosis involving parietal pericardial fat are reported.

2. These cases, to the best of our knowledge, represent a previously undescribed clinicopathologic entity.

3. All patients presented clinically with acute anterior chest pain, and their thoracic roentgenograms demonstrated a lesion in juxtaposition to the heart.

4. Histologically, the surgical specimens demonstrated the usual picture of fat necrosis and, in addition, abnormalities of some of the blood vessels.

5. These vascular changes were demonstrated in fat necrosis of other sites and are believed to be secondary in nature.

A moderate degree of inflammatory reaction enveloped the mass, but the lobes were separated from it without damage. It was then recognized as a large necrotic fat pad and was removed from the parietal pericardium without difficulty.

The involved fat (Fig. 6) measured 7.4 by 6 by 2.5 cm. It was firm and again appeared browner than the surrounding fat. Microscopically it revealed the changes of fat necrosis and was not unlike the fatty masses in Cases 1 and 2. The inflammatory reaction was mild, with a moderate amount of fibrosis and a very extensive degree of phagocytosis of fat. Some of the small veins and arteries showed rather marked endarteritic changes, with considerable narrowing of the lumina. Proliferation of intimal cells and subintimal fibrosis were demonstrated.

## COMMENT

The similarity of clinical findings in the foregoing patients is at once obvious. Onset of difficulty in each was heralded by acute low anterior chest pain, and thoracic roentgenograms demonstrated a mass close to the cardiac silhouette. All had a pleuritic component to their pain at some phase of their illness, and in 2 it was referred to the shoulder. In general, symptoms persisted, to become chronic, although intermittent in character. Each patient was quite obese, and we are inclined to believe that this is probably a prerequisite of their disease. In retrospect, then, these findings appear to be sufficiently constant to warrant clinical consideration of the correct diagnosis in any future case. Proper therapy would, however, indicate thoracotomy with resection of the diseased tissue. On surgical exposure, the true nature of these lesions may not be immediately evident, since they are enveloped by a moderately intense secondary inflammatory reaction. On cut surface, however, they are composed entirely of fat and are readily recognized as such.

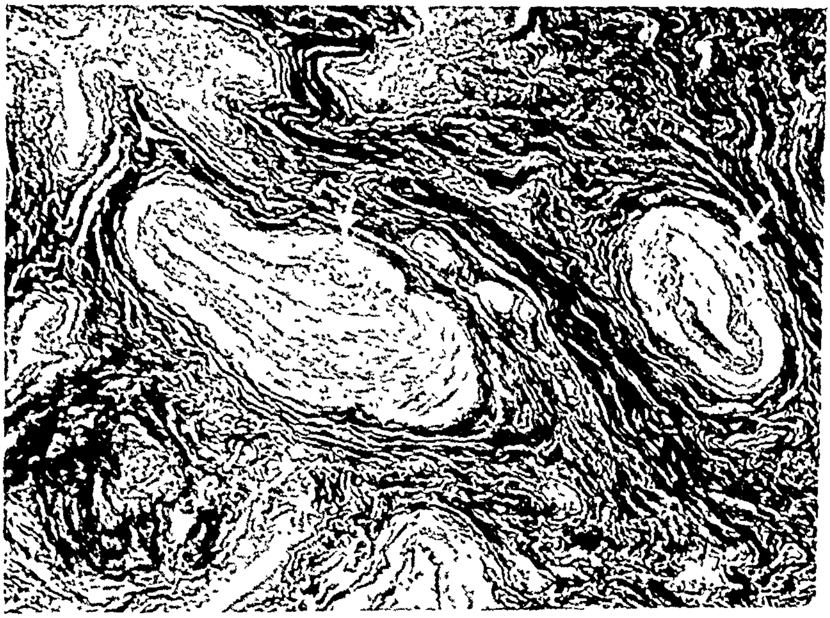


Fig. 7.—Section from a surgical specimen of epiploic appendicitis, showing subintimal fibrosis of the arterioles (arrows) (elastic van Gieson; ×90).

In 2 of the patients reported in this paper the acute attack of anterior chest pain suggested the diagnosis of myocardial infarction. This diagnosis

*Past History*.—The past history did not contain additionally helpful data. There was no history of tuberculous infection or of contact with tuberculosis.

*Physical Examination*.—The patient was acutely ill with obvious respiratory distress. He was also emaciated. The temperature was 100.2° F. and pulse was 128 per minute. Respiratory rate was 26 per minute. The neck veins were markedly distended. Two ulcers were noted near the center of a roughly oval area of induration on the left lateral chest wall. The indurated area measured 8 cm. in greatest diameter and was near the mid-axillary line overlying the left ninth, tenth, and eleventh ribs. Another area of induration (4 cm. in greatest diameter) was noted in the left midclavicular line overlying the fifth and sixth ribs. This lesion was tender but was not ulcerated.

There was dullness to percussion in the lower one-half of the left chest. In this region breath sounds were absent, although elsewhere coarse râles were heard in both lung fields. Respiratory excursion of the left hemithorax was markedly diminished and there was no visible or palpable evidence of cardiac pulsation in the left precordium. The heart sounds were distant and no murmur was heard. Blood pressure was 108/92 mm. Hg. A paradoxical pulse was present which encompassed the entire range of pulse pressure. The abdomen was ovoid, protuberant, and tense. Shifting dullness to percussion was noted and the liver edge was palpable 10 cm. below the right costal margin. Massive dependent edema was present which involved both lower extremities and extended up the trunk to the level of the heart.

*Bacteriologic Data*.—Typical "sulfur granules" were identified in purulent material draining from the left chest wall. Clubbing was seen at the periphery of these granules. Further examination of a crushed granule revealed numerous gram-positive branching filaments which resembled an actinomycete. The organism was not acid fast with 3 per cent acid alcohol decolorization. It was cultured on blood agar in a "Brewer anaerobic jar" where rough colonies resembling "bread crumbs" appeared after 4 days' incubation at 37° C. Examination of smears from the colonies revealed morphologic and staining characteristics similar to those just described. This organism was identified as *Actinomyces bovis*.

*Laboratory Data*.—Hemoglobin was 7.5 Gm. per 100 c.c., hematocrit 25 per cent, red blood cells, 3.5 million per cubic millimeter, white blood cells, 5,400 per cubic millimeter, and differential count showed 87 per cent polymorphonuclear leukocytes. Urine specific gravity was 1.002. Tests for urine sugar and albumin were negative. Nonprotein nitrogen was 22.5 mg. per cent. Total protein was 5.78 Gm. per cent with 3.52 Gm. albumin, and 2.26 Gm. globulin.

Examination of the posteroanterior chest x-ray revealed marked enlargement of the cardiopericardial silhouette. Irregular areas of increased density were present in the left lower lung field extending from the level of the diaphragm to the anterior portion of the left fourth rib. The area of greatest density measured 1.5 by 3 cm. and was adjacent to the lateral chest wall corresponding in position to the larger region of induration described above. The right costophrenic angle was blunt. The electrocardiogram showed low QRS voltage with negative T waves in Leads I, II, III, $V_3$, and $V_5$ (Fig. 1). Venous pressure was 280 mm. of water and arm-to-tongue circulation time (Decholin) was 13 seconds. The hepatojugular reflex phenomenon was markedly positive.

*Hospital Course*.—Following admission the fever, restlessness, marked dyspnea, and orthopnea continued. He was given a low sodium diet (200 mg. per day) and advised to rest in bed. The day after admission the following therapy was instituted: intramuscular aqueous crystalline penicillin (4 million units daily), oral sulfadiazine (4 Gm. daily), and oral tetracycline (1 Gm. daily). Penicillin was changed to intramuscular aqueous procaine penicillin (1.2 million units daily) after three days of therapy. His temperature returned to normal five days after the initiation of treatment. By this time dyspnea and orthopnea decreased in severity, but hepatomegaly, ascites, and ankle edema persisted. Further radiographic studies, including roentgenkymography showed minimal to absent

# CHRONIC CONSTRICTIVE PERICARDITIS DUE TO ACTINOMYCES BOVIS

## Report of a Case Treated by Pericardectomy

M. Hara, M.D., and J. A. Pierce, M.D.
Little Rock, Ark.

ACTINOMYCES BOVIS is an uncommon cause of chronic constrictive pericarditis. This occurrence is not surprising in view of the pathologic behavior of this fungus which resembles that of the tubercle bacillus in certain respects. Of the 71 cases of actinomycosis of the heart and pericardium reported in the world literature since 1882, approximately 14 patients have presented the clinical syndrome of chronic cardiac constriction.

Prior to the advent of effective therapy, involvement of the heart or its membrane had occurred mainly as a late or terminal complication of a lethal infection. Consequently, the uncontrollable and diffuse nature of the infection in chronic obliterative pericarditis had uniformly precluded treatment by pericardectomy. More recently the beneficial action of the sulfonamides and penicillin has largely prevented the complication of pericardial and cardiac actinomycosis and permitted the safe undertaking of operative procedures in patients. This communication concerns a patient with chronic constrictive pericarditis due to *Actinomyces bovis* who is felt to represent the first documented instance in which pericardectomy has been carried out successfully.

### CASE REPORT

*Present Illness.*—A 35-year-old Negro man, unit number A-72209, was admitted to University Hospital on Jan. 7, 1955, complaining of dyspnea, abdominal swelling, and ankle edema of four weeks' duration. He had been well until five months previously when he noted an area of tenderness on the lower left chest wall laterally. This area became indurated, softened centrally, and ruptured after several days. A small amount of drainage appeared which persisted for a few days following which the lesion gradually healed. He had no further symptoms until five weeks before admission when tenderness and induration reappeared in the same region. Within several days, this lesion ruptured and drained.

Scanty purulent drainage persisted and one week later (four weeks prior to admission) the patient developed severe dyspnea. He soon noticed abdominal swelling and ankle edema. He stated his heart felt "swollen." The dyspnea became increasingly severe, and orthopnea developed so that on three or four occasions he sat in a chair all night. Fever, night sweats, and frequent headaches were troublesome, and later he developed a cough which was productive of a small amount of mucoid sputum. The headache abated one week prior to admission, but the other symptoms persisted.

---

From the Department of Surgery and the Department of Medicine, University of Arkansas School of Medicine, Little Rock, Ark.

Received for publication May 14, 1956.

*Pathologic data:* Grossly, the pericardium was purplish-tan in color and varied from 3 to 10 mm. in thickness. Examination of material, obtained by sectioning the specimen, revealed gram-positive branching filaments, but cultures of this material failed to grow. Sections were prepared with hematoxylin-eosin, periodic acid-Schiff reagent, and Ziehl-Neelsen acid-fast stains.

Microscopic examination revealed a markedly thickened pericardium composed of fibrous tissue. A few sharply defined collections of polymorphonuclear leukocytes were seen which were considered typical micro-abscesses. One colony of *Actinomyces bovis* was found in such a lesion (Fig. 3). Numerous small blood vessels were present in the pericardium and, perivascularly, lymphocytic infiltration and scattered hemorrhages were noted. Some focal areas of round-cell infiltration were also present. No acid-fast organisms could be demonstrated. The final pathologic diagnosis was chronic constrictive pericarditis due to *Actinomyces bovis.*

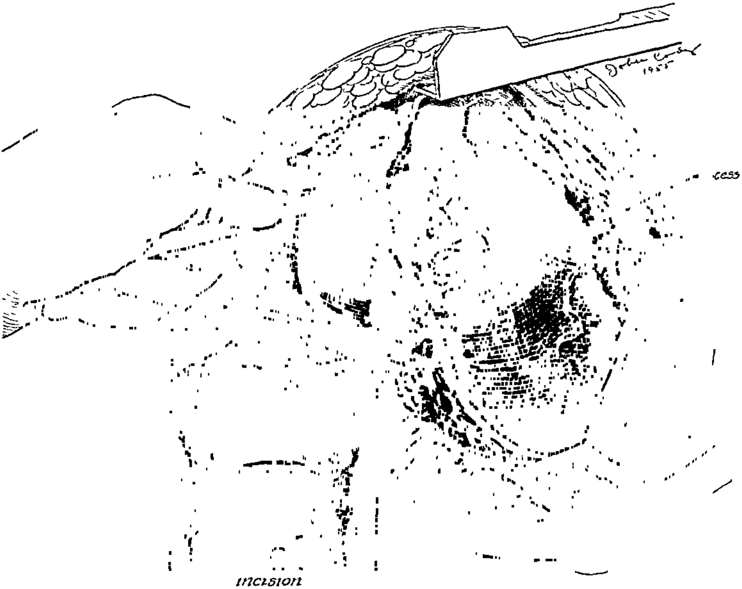


Fig. 2.—Artist's drawing of operative findings.

*Postoperative course:* His recovery following surgery was uneventful. The day after operation venous pressure was 146 mm. of water and arm-to-tongue (Decholin) circulation time was 19 seconds. He had a low-grade fever for six days although he reported subjective improvement. Subsequent determinations of venous pressure and circulation time were repeatedly within normal range. He was released from the hospital completely free of symptoms on March 26, 1955.

Following discharge he continued to receive intramuscular aqueous procaine penicillin (1.2 million units daily) and oral sulfadiazine (3 Gm. daily). He was free of symptoms when last seen, three months postoperatively. Examination did not reveal hepatomegaly or ankle edema. He was advised to continue medication for three additional months.

cardiac pulsations. Pericardiocentesis was attempted from three different approaches but was unsuccessful. During the latter part of February, cardiac insufficiency was increasingly severe as evidenced by an increasing venous hypertension (maximum 380 mm. water) and marked prolongation of the circulation (maximum 43 seconds). Because of this, operative intervention was advised.

*Operation.—Operative findings:* An abscess, 3 cm. in diameter, containing minute particles was found in the soft tissues just anterior to the left fifth rib, 8 to 10 cm. from the costochondral junction. The heart, which was not enlarged, was encased in a thick fibrous capsule which markedly restricted its activity. The membrane measured 3 to 10 mm. in thickness. No exudate or fluid was encountered during decortication of the heart which was carried out with relative ease.

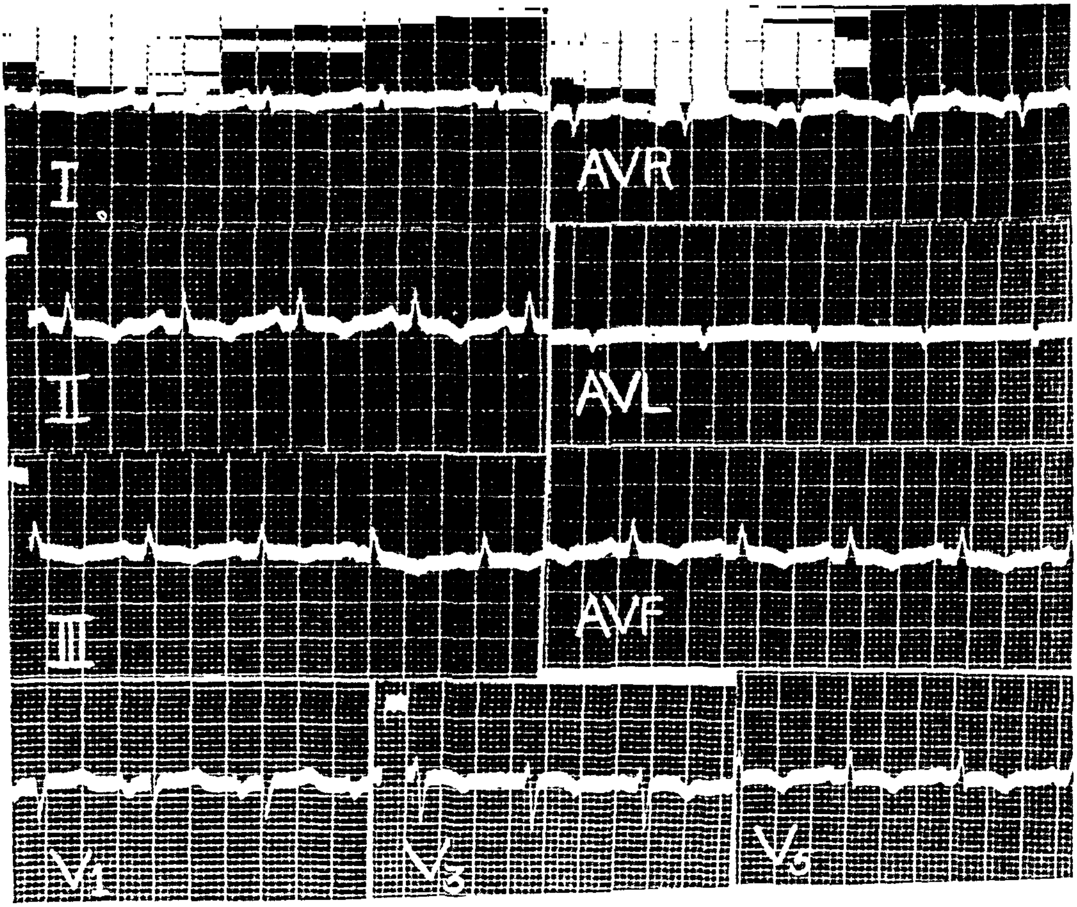


Fig. 1.—Electrocardiogram showing changes typical of pericarditis. (See text.)

*Operative procedure:* A transverse sternum-splitting incision was made in the fifth intercostal space. The upper end of the sternum was then split vertically 6 cm. The thickened membrane was removed from both ventricles anteriorly, down to the myocardium, carrying the excision 2.5 cm. posterior to the left phrenic nerve. Inferiorly, the entire diaphragmatic aspect of the heart was liberated and, superiorly, the atrioventricular groove on both sides was exposed. The heart action improved dramatically as the decortication progressed and became strong and vigorous after removal of the peel. The pulse rate and blood pressure were stable throughout the procedure. Closure of the wound was accomplished with silk sutures.

by actinomycotic granulation tissue. In those remaining, the changes ranged from mild inflammation confined to the parietal pericardium to a more severe fibrinopurulent process. In one patient, 750 c.c. of pus was recovered. In 20 of the 29 patients with proved pericarditis, invasion of the myocardium had occurred, leading to the formation of multiple abscesses with frequent extension of the process to the endocardium. At times the granulomatous masses actually projected into the cardiac chamber with perforation of the endocardium and pyemia in 6 patients.

Cornell and Shookhoff[1] noted that only 23 of 68 patients were reported to have manifested clinical evidence of disease of the heart or its membranes. However, the lack of data did not permit an accurate definition of the clinical picture in a significant number of the patients. The patients reported by Gose[2] and Lidbeck,[3] both of whom have been mentioned but not reviewed by Cornell and Shookhoff, were also shown to have chronic constrictive pericarditis. The syndrome of chronic constrictive pericarditis was absent in the others with demonstrable generalized obliterative pericarditis at necropsy. That an adherent pericardium is not necessarily synonymous with clinical cardiac constriction has been borne out by the study of Sprague, Burch, and White.[4] Their review of 43 patients with adherent pericardium among 1,900 autopsies at the Massachusetts General Hospital failed to disclose a single example of clinical constrictive pericarditis.

In the ten years since the report of Cornell and Shookhoff, no additional patients with chronic constrictive pericarditis due to *Actinomyces bovis* have been recorded in the literature. Zoeckler[5] has reported a patient with actinomycotic pericarditis in whom the chief clinical manifestation was the presence of a pericardial friction rub. Treatment with penicillin and Aureomycin resulted in complete recovery without sequelae. The rare occurrence of cardiac or pericardial actinomycosis during the past decade can undoubtedly be attributed to the efficacy of the antibiotic drugs in eradicating this infection.

The term "actinomycosis" is considered by Conant and Rosebury,[6] and others,[7, 8] as referring specifically to infections caused by *Actinomyces bovis*. Publications which do not conform to this terminology have not been considered in the present report. In this connection, it has not been possible to determine how many of the early reports dealt with infections caused by species other than *Actinomyces bovis*. The colony of organisms seen in Fig. 3 was not observed on routine histologic examination, but was discovered only after a large number of microscopic sections had been examined. This fact points out that actinomycotic granules might be easily overlooked in the case of healing pericarditis. Attempts to culture the organism at the time of surgery were unsuccessful although its staining characteristics were similar to those observed before treatment.

The role of surgery in diffuse adherent pericarditis due to the ray fungus has been a singularly insignificant one, a fact attributed to the ineffective control of the infection prior to the availability of present day therapy. As far as it can be ascertained from the available literature, pericardial decortication has been attempted only once prior to that of our patient. He was a

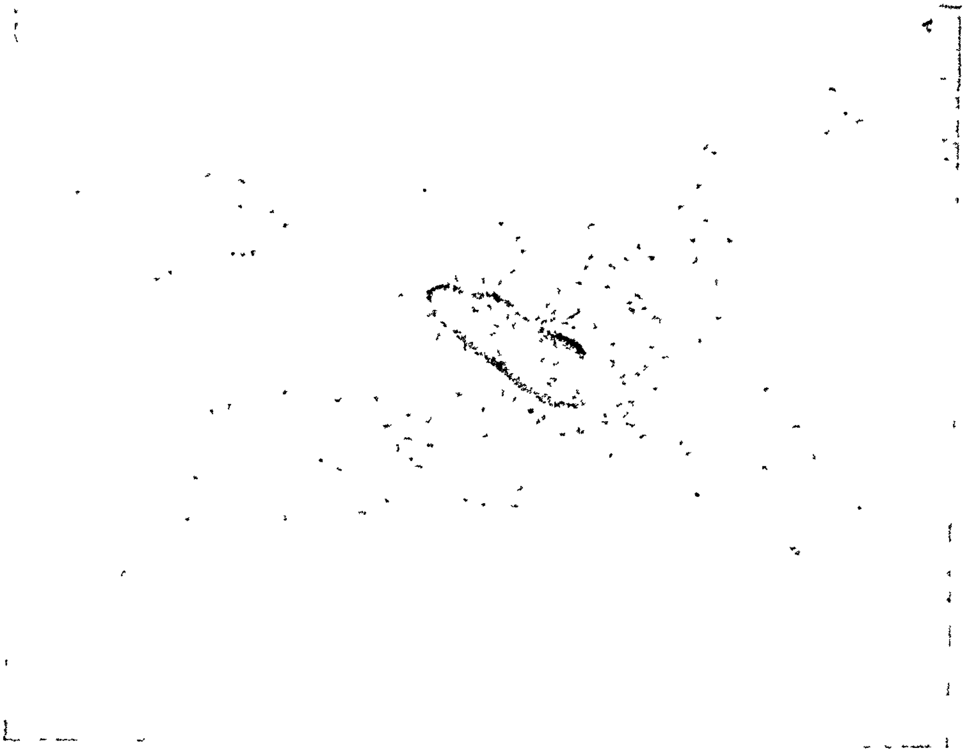


Fig. 3.—Photomicrograph of pericardium containing a typical "sulfur granule." Periodic acid-Schiff stain. (×400.)

## DISCUSSION

The pathogenesis of actinomycotic pericarditis is discussed by Cornell and Shookhoff[1] who reviewed, in 1944, the clinical and pathologic findings in 68 patients, including 3 of their own with actinomycosis of the heart and/or pericardium collected from the world literature. The cases of 2 additional patients with this lesion have been appended to the end of their article, making a total of 70 patients.

The pathologic process observed at necropsy in 69 was similar to that noted in actinomycosis of other organs and tissues, characterized by a granulomatous reaction upon which are superimposed the features of acute and chronic suppuration. Microscopically, the lesions consist of minute abscesses surrounded by an actively proliferating connective tissue in which are scattered polymorphonuclear leukocytes, epithelioid cells, and numerous capillaries.

The pericardium was found to be most commonly involved by direct spread from a contiguous focus, most frequently in the lungs. Occasionally the pericarditis was a sequela of active infection within the myocardium which in turn had resulted from blood stream infection. Forty-one patients of the collected group studied by the authors fell into the former category, although in 12 patients the organism could not be demonstrated in the pericardial tissue. However, in each of the latter instances, a focus of actinomycosis was proved to be present adjacent to the heart. In 18 of 38 cases in which sufficient data were available the pericardial sac was completely obliterated

## SUMMARY

1. A patient is presented with chronic constrictive pericarditis caused by *Actinomyces bovis.*

2. The principles of therapeutic management were essentially those followed in the treatment of constrictive pericarditis of tuberculous etiology.

3. This patient is thought to represent the first reported instance of successful pericardectomy in actinomycotic constrictive pericarditis.

## REFERENCES

1. Cornell, A., and Shookhoff, H. B.: Actinomycosis of the Heart Simulating Rheumatic Fever, Arch. Int. Med. 74: 11, 1944.
2. Gose, A. C.: A Case of Constrictive Pericarditis Due to Actinomycosis, Memphis M. J. 17: 56, 1942.
3. Lidbeck, W.: Actinomycosis: Report of Two Cases Showing Uncommon Clinical and Pathological Features, West. J. Surg. 50: 498, 1942.
4. Sprague, H. B., Burch, H. A., and White, P. D.: Adherent Pericardium and Pick's Syndrome, New England J. Med. 207: 483, 1932.
5. Zoeckler, S. J.: Cardiac Actinomycosis: A Case Report and Survey of the Literature, Circulation 3: 854, 1951.
6. Conant, N. F., and Rosebury, T.: The Actinomycetes, in Dubos' Bacterial and Mycotic Infections of Man, Philadelphia, 1952, J. B. Lippincott Co., p. 634.
7. Smith, D. T.: Therapy for the Systemic Fungous Infections, New York State J. Med. 54: 2303, 1954.
8. Martin, D. S.: Actinomycosis in Harrison's Principles of Internal Medicine, New York, 1954, The Blakiston Co., p. 983.
9. Bigland, A. D., and Sergeant, C. H.: A Case of Actinomycosis With Recovery, Brit. M. J. 2: 61, 1923.
10. Johnson, J., and Kirby, C. K.: A New Incision for Pericardectomy, Ann. Surg. 133: 540, 1951.

53-year-old white man, reported by Gose in 1942, who developed the characteristic picture of chronic constrictive pericarditis while he was under treatment for an undiagnosed infectious process in the right lower lobe. His course was extremely septic and progressively downhill. Pericardectomy was recommended for relief of his cardiac symptoms despite the continuing deterioration of the patient. At thoracotomy, the surgeon discovered an inflamed and thickened pericardium, and upon excising a portion of the parietal pericardium overlying the left anterior atrioventricular region, uncovered a large abscess. The operation was terminated at this point. The patient died on the day following surgery. Necropsy revealed a greatly thickened and indurated pericardium with sinuses and obliteration of the sac except for the previously described abscess. The underlying myocardium was not involved. Actinomyces were isolated in the exudate from the pericardium and from the operative incision, but were not cultured. The source of the pericarditis was traced to an abscess in the left lung.

The patient reported by Bigland and Sergeant,[9] in 1923, is the sole survivor in the collected series prior to Zoeckler's patient and deserves brief comment. This 22-year-old white man developed a massive purulent pericardial effusion due to *Actinomyces bovis.* Pericardiostomy, performed as a desperate measure, saved his life. He had been treated initially for postpneumonic empyema of the right pleural cavity by a rib resection but failed to improve satisfactorily. His dyspnea became ominously intense. A teleoroentgenogram of the chest revealed opacification of the left lung to the level of the sixth rib. After a pericardiocentesis was unsuccessful, incision into the pericardium yielded 25 ounces of a seropurulent fluid. The pericardial surface was shaggy. The patient recovered after a stormy convalescence without the sequela of chronic constrictive pericarditis. Attempts to demonstrate organisms from the original fluid from both the empyema cavity and the pericardial sac failed, but Actinomyces were later isolated in the drainage from the chest cavity.

The clinical problems and gross pathologic findings in our patient did not differ essentially from those frequently seen in patients with tuberculous chronic constrictive pericarditis. Although the surgical experience in actinomycotic pericarditis is meager, the principles commonly advocated in the surgical management of tuberculous constrictive pericarditis are equally applicable. Although the transverse sternum-splitting approach, as utilized by Johnson and Kirby,[10] and others, gave excellent exposure of the heart, the placement of the incision through the site of the healed sinus tract resulted in the inadvertent opening of an abscess in the chest wall. Fortunately, this did not lead to infection of the wound or other harmful complications. The alternative and better incision in this case would have been a medium sternotomy to circumvent completely the region of the abscess. The outcome in this patient would indicate that pericardial decortication can be safely performed relatively soon after subsidence of an active infection.

have recurred and there is ever present the danger of the inhalation of stomach contents into the lungs during sleep, but this method of treatment is undoubtedly a great advance on anything that has been done before.

In 1953, Brain[1] published some results on fat absorption after various gastric operations, and on these findings recommended the excision of esophageal peptic strictures, the reduction of the stomach into the abdomen and restoration of continuity by an isolated intestinal loop within the mediastinum and abdomen. This was a modern adaptation within the mediastinum of what Roux[2] had originally performed beneath the skin of the chest wall. In the same paper, Brain gave an account of the one patient on whom he had performed the operation successfully in November, 1951, and he now reports in a personal communication that he has done three more. If this operation could be done safely it would seem to provide a satisfactory answer to the problem in children and might even be more generally applicable. The possible objections to the operation are first that it involves many anastomoses and that there are therefore many opportunities for technical mistakes, second that ulceration might occur at the jejunogastric junction, and third that there might be difficulty in vomiting should the need arise. The first of these listed dangers inevitably persists as the following records will show. The earliest patient operated upon in the series died as a result of technical faults, and the description of many other mistakes that have been made and then corrected may be a help to other surgeons working in this field. No gastrojejunal ulceration has yet been observed, and one child who ate too much rich food vomited successfully.

The technique of esophagojejunogastrostomy is here described and some details of the 11 patients upon whom it has been performed are recorded.

*Operation.*—A left thoraco-abdominal incision is made through the eighth space and the diaphragm is split back toward, but not into, the hiatus. The edges of the diaphragm are conveniently retracted by being sutured to the edges of the costal incision. The gastrosplenic omentum is divided, the vasa brevia being ligatured, so that the fundus of the stomach and the left half of the greater curvature are free. The stomach is rotated forward and the vessels at the upper end of the lesser curvature secured and divided from the back. The stomach is divided just below the cardia and the two ends sutured and infolded. The cardia is drawn up into the mediastinum and the diseased part of the esophagus is dissected free with a length of normal esophagus above, to give both space and mobility for an easy anastomosis. The vagus nerves may be dissected free from the wall of the esophagus in the hope of preserving some of their fibers, but it usually happens that most of them are divided before the operation is completed. The esophagus is then allowed to "lie fallow" in the mediastinum, while a Roux loop, long enough to reach the normal part of the esophagus without tension, is fashioned. The details of construction of a Roux loop have already been described (Allison and Da Silva[3]) and do not differ materially from that in the original paper by Roux,[2] himself (1907). The upper end of the loop is closed by inversion and suture.

# ESOPHAGOJEJUNOGASTROSTOMY

P. R. ALLISON, F.R.C.S., F.A.C.S.(HON.), OXFORD, ENGLAND, G. H. WOOLER, F.R.C.S., LEEDS, ENGLAND, AND A. J. GUNNING, F.R.C.S., OXFORD, ENGLAND

THE performing of esophagojejunostomy by Roux loop, after radical total gastrectomy for malignant disease of the stomach, provided enough technical experience for the method to be applied to intestinal reconstruction after the removal of simple peptic strictures of the esophagus. A regular survey of those patients who survived for years after excision of a carcinoma showed that, with occasional medical care, their weight, strength, and capacity for work were much better than had been anticipated; some of them are still working and taking a normal diet five to eight years after operation. When esophagojejunostomy was used for benign lesions of the esophagus, it was usually unnecessary to resect the stomach. This was, therefore, closed at its upper end and left in place as a blind appendage to the alimentary canal. Food then passed from the esophagus into the jejunum where it travelled for twelve to eighteen inches before it met the bile and pancreatic juice and such gastric juice as was secreted. The patients with benign lesions form an interesting group for study, for they should have a long expectation of life.

Groups of patients, 26 who have had radical resections for malignant disease and survived more than 3 years, 10 with total resections for benign lesions (some of them thought to be malignant at operation), and 42 patients who had had esophagojejunostomies for peptic stenosis of the esophagus (in most of whom the stomach was left in place), have formed a modest basis on which to assess the effects of such drastic alterations to the alimentary tract on nutrition, strength, and habits of life. The patients were mostly past middle age and their progress over the years will be the subject of a further report. The results have been generally good, some have been excellent and some very poor, a description that might be applied with equal validity to almost any operation on the stomach that alters the design of nature. However encouraging some of these results might be in middle-aged and elderly people, they hardly seemed to justify doing the same operation on a small child. The idea of by-passing the stomach and duodenum in a child 4 or 5 years old with its special needs for growth, development, maturation, and reproduction was inadmissible, and yet these were the patients who seemed to be most urgently in need of help. Some surgeons have tackled the problem by performing long esophageal resections and high mediastinal esophagogastrostomy. This operation has been immediately effective in relieving the dysphagia, but it will be some time before the metabolic results become apparent. Some strictures

---

Received for publication May 8, 1956.

and it is therefore left draining into a bottle for the first few days after operation. Apart from allowing for the escape of gas it is also very useful for determining the state of intestinal and gastric motility, because when the patient is fed by mouth the time of arrival of food in the stomach can be observed, and by closing the tube for a short time after a meal and then opening it again the rate of stomach emptying can be estimated and so the timing of feeds controlled. Another advantage of a gastrostomy is that the patient can be given drinks as soon as he recovers from his anesthetic. The effect of being able to drink by mouth is a great relief and encouragement to a patient, even if the fluid escapes through a gastrostomy drain. His fluid requirements in these early days are maintained by intravenous infusion.

The following case histories are given, not only to illustrate the ultimate efficiency of the operation but to describe the many mistakes that have been made.

## CASE REPORTS

CASE 1.—P. B., a male child, was aged 2½ years when first admitted to the hospital, had vomited since birth and had never been able to take solid food. No tumor had ever been felt at the pylorus. Radiography showed persistent narrowing of the junction of the middle and lower thirds of the esophagus and retention of barium in the stomach five hours after ingestion. Esophagoscopy confirmed the presence of a stricture with apparent periesophageal induration at a level estimated to be just below the bifurcation of the trachea. Over the succeeding ten years the child was esophagoscoped and gently dilated ten times. By then the esophagus was stenosed over a distance of 7 cm., the stomach was large with poor peristalsis, but no abnormality could be detected in the pylorus or duodenum.

Operation (P. R. A.) was performed on Dec. 8, 1953, as already described, except that no pyloromyotomy was done. There was no evidence of ulcer or scar at the pylorus. Eight arteriae rectae were divided and 25 cm. of intestine was resected below the level of the jejunogastric anastomosis. An end to end entero anastomosis was performed below the transverse mesocolon. The boy developed obstruction at this anastomosis and was operated upon twice for its relief, but the loop in the chest became distended and perforated to form an empyema. During an operation for this last condition, he inhaled pus and barium and died shortly afterward of bronchopneumonia.

CASE 2.—B. M., a male child, was first admitted to the hospital at the age of 13 months because of vomiting since birth and inability to take solid food. No pyloric tumor could be felt. Radiography showed a stenosis of the distal part of the esophagus with sliding hiatal hernia (Fig. 1), a large stomach, and a deformity of the duodenum. Esophagoscopy confirmed the presence of a tight peptic stenosis. During the succeeding seven years the boy was esophagoscoped eight times.

Operation (P. R. A.) was performed on Feb. 17, 1953. The cardia was 2 cm. above the hiatus and the stenosis of the esophagus extended from here up to 2 cm. below the aortic arch. Resection and esophagojejunostomy were performed at the level of the aortic arch (Fig. 2). The loop was transected below and the end implanted into the posterior aspect of the stomach in the region of the cut vasa brevia (Fig. 3). Five arteriae rectae were divided and the remaining part of the loop turned down and anastomosed, end to end, to the segment of jejunum descending from the duodenum. Neither a gastrostomy nor a pyloromyotomy was performed. Three days after operation, the patient became very ill with abdominal cramps, distention of the abdomen, and dyspnea. The abdomen was opened and an enormously distended stomach was found to be filling the upper abdomen. It was decompressed slowly by the withdrawal of 3 pints of watery and rather bile stained

At this point it is useful to perform a pyloromyotomy, for the stomach is still free and a choice can therefore be made between the anterior and posterior surface of the pylorus, depending on which is the more accessible. Care is needed if the muscle fibers of the pylorus are to be completely divided without injury to the duodenal mucosa, for, since the pylorus projects into the duodenum, the mucosa of the latter comes to overlay the deepest fibers of the muscle and is easily opened (by mistake) before all the muscle has been separated. From the experience so far gained, the performance of pyloromyotomy is considered to be a useful safeguard against gastric delay and distention in the weeks following operation when the peristalsis of the stomach is weakened by vagal paralysis.

A hole is next made in the transverse mesocolon, as far back as possible on the posterior abdominal wall so that no ridge occurs here when the transverse colon is drawn down. Through this hole, the Roux loop is threaded and passed up behind the stomach in the now widely opened lesser sac of peritoneum, through the hiatus and into the mediastinum. The hiatus may be enlarged a little by stretching its walls so that the loop is not held tightly. The subterminal part of the loop is joined to the musculofascial layer of the esophagus above the stricture, the diseased part of the esophagus excised, and the anastomosis completed. The loop below the diaphragm is so arranged that it passes over to the left behind the fundus of the stomach and reaches the greater curvature in the region of the gastrosplenic omentum, and a point is selected on the loop that can be anastomosed to the posterior surface of the stomach without tension or kinking. The intestine is then divided at this chosen point. One or two of the arteriae rectae of the upper segment are divided in order to free the circumference of the intestine and so facilitate the anastomosis to the stomach. The intestine below the level of the section must then be freed so that it can be drawn down and reunited to the small length of jejunum attached to the duodenum below the transverse mesocolon. This may mean the division of six to ten arteriae rectae and even the removal of a short segment of the intestine itself. The upper segment of the loop is anastomosed end-to-side to the posterior aspect of the stomach. For this purpose, it is probably wise to cut the end of the intestine obliquely or "on the bevel" so that it is not made to kink by the necessity of turning abruptly forward to join the stomach. The lower segment is joined to that piece of jejunum left in continuity with the duodenum by as long a side-to-side anastomosis as possible. The hole in the transverse mesocolon, which now only transmits the mesentery carrying the blood vessels of the isolated loop, is closed above the duodenojejunal flexure and above the entero-anastomosis. The stomach is stitched back to the diaphragm to remake the lesser sac of peritoneum and a gastrostomy is performed in its anterior surface. After this operation, patients always swallow air and, whereas, in the absence of a pyloromyotomy the stomach becomes inflated, in its presence, the air passes on into the intestines and causes generalized distention. The performance of a gastrostomy is not for the purpose of feeding but to allow the escape of gas, Case 4—J G, a man aged 34 years, had had vomiting since birth and dysphagia for solid food since the first time it was tried He was underweight and underdeveloped, had never had a solid meal in his life, and had never been able to take part in "social" eating Radiography showed a tight stricture with an ulcer crater or diverticulum at a level 5 cm below the bifurcation of the trachea Below this was a segment that might have been esophagus lined with gastric mucosa and then a sliding hiatal hernia The upper segment of the esophagus was dilated and showed very vigorous peristalsis after swallowing There was no abnormality at the pylorus A tight stenosis, at 36 cm, was confirmed by esophagoscopy but no view was obtained beyond it

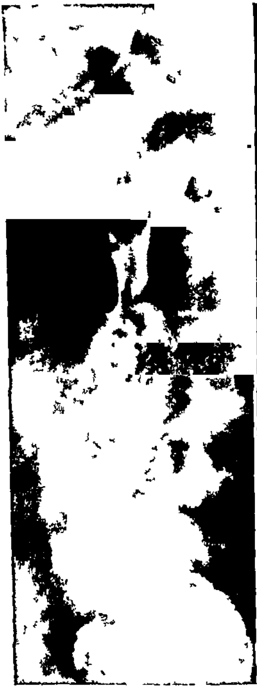


Fig 2

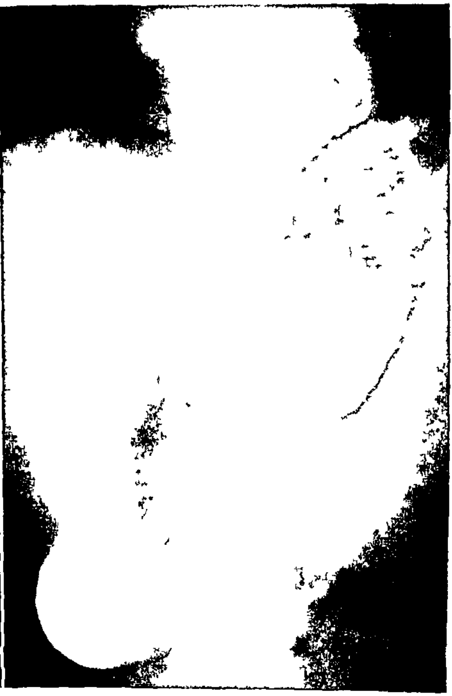


Fig 3

Fig 2—Case 2 Roux loop anastomosed to esophagus at the level of the aortic arch after excision of peptic stenosis

Fig 3—Case 2 The lower end of the isolated Roux loop anastomosed to posterior surface of the stomach in the region of the cut vasa brevia

Operation (P R A) was performed on Oct 7, 1953 The vascular arcades of the small intestine were very bizarre so that the effective length of the Roux loop was small in relation to the length of jejunum composing the loop The spleen was three times it normal size and was some impediment to the isolating and severing of the cardia The esophagus, in the region of the stricture, had to be freed from the pericardium by knife dissection The esophagojejunal anastomosis was made and eight arteriae rectae divided fluid. The duodenum and the jejunum, for 10 cm. beyond the entero anastomosis, were also distended. The obstruction was apparently caused by the kinking and adhesion of the intestine at this level. The adhesions were divided, the gut straightened, and a gastrostomy performed.

Two years later the boy was seen to have developed well, was eating "anything and everything," and had no complaints.

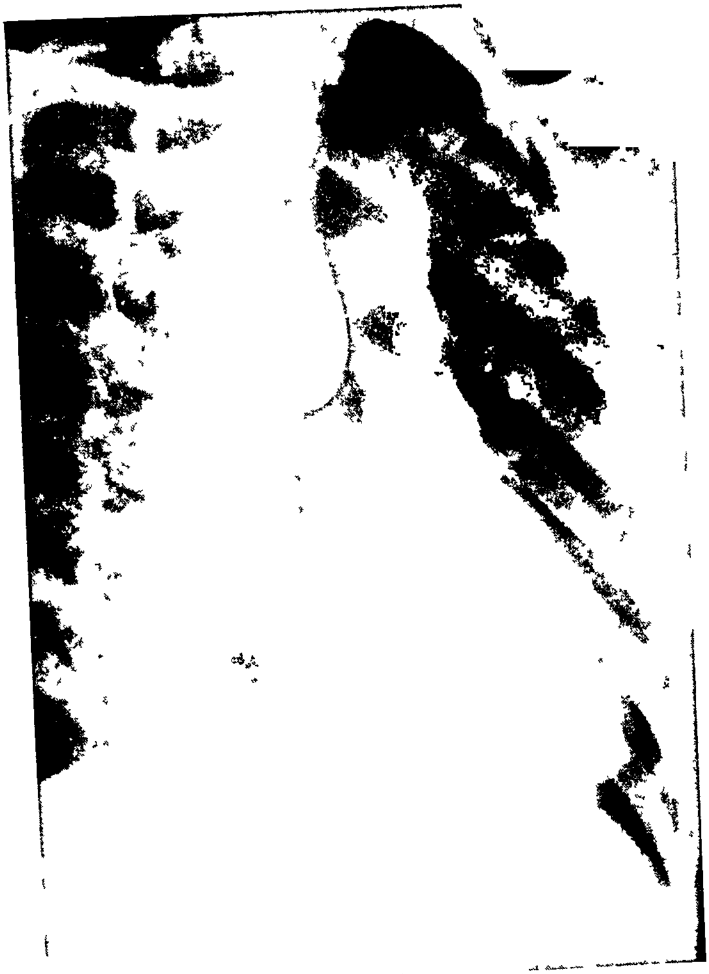


Fig 1—Case 2 Long peptic stenosis of esophagus with sliding hiatal hernia

CASE 3.—A. P., a man aged 66 years, suffered for one year from dysphagia and epigastric pain. Radiography showed an irregular stenosis 5 cm. above the cardia and esophagoscopy confirmed that this was a simple peptic ulcer.

Operation (A. G. Morrow) was performed on May 14, 1953. This was carried out as described but no pyloromyotomy was done, and intestinal continuity was restored by end-to end anastomosis. The loop between the esophagus and stomach was redundant and became kinked and dilated in the chest. In addition, there developed some narrowing of the esophagojejunal stoma The wound was reopened about five weeks after the first operation, the anastomosis and the redundant loop were resected and a new anastomosis made.

The patient's progress was satisfactory and, when last seen, two years after operation, he was eating normally, had put on weight, had a normal blood count, and was free from symptoms. Barium passed through all segments of the alimentary canal normally.

Operation (P R A ) was performed on Feb 9, 1954 Esophagus lined by gastric mucosa was found between the stricture and the herniated stomach The resection and anastomosis were carried out as described, pyloromyotomy and gastrostomy were done, and intestinal continuity was restored by a large side to side anastomosis

Recovery was without incident, and eighteen months later the patient was eating everything that was put before him, was developing rapidly, and was well in every way

CASE 7 —R C, a woman aged 54 years, had had dysphagia for solid food for five years Radiography and esophagoscopy showed that she had a peptic stenosis of the esophagus with a sliding hiatal hernia

Operation (P R A ) was performed on Aug 7, 1955 This was just as described Pyloromyotomy, gastrostomy, and side to side entero anastomosis were used Recovery was uneventful and the patient has been completely free from symptoms and eating a normal diet to the present time

CASE 8 —J G, a woman of 37 years, had vomited since the eighth day of life and had never been able to eat solid food She had had various forms of medical and psychologic treatment and many years of bougienage The esophagus had been perforated by a bougie with a resulting right hemothorax and at that time a gastrostomy was done Some years after this, the peptic stenosis of the esophagus had been resected, esophagogastrostomy performed, and the gastrostomy allowed to heal A further peptic stenosis had developed, and complete collapse of the right lung Two separate plastic operations had been done to open up the stenosis but these had given only very temporary relief and a gastrostomy had to be remade to save the patient's life Esophagoscopy showed a complicated and ulcerated stricture with excessive granulation tissue at 32 cm

Operation (P R A ) was performed on Aug 16, 1955 The old scars were excised The lesser sac was opened and a pyloromyotomy performed on the posterior aspect to avoid disturbing the gastrostomy The esophagogastrostomy was freed and resected, the stomach closed and returned to the abdomen The rest of the operation was as described In the postoperative period there was some obstruction by kinking of the loop at the jejunogastric junction The kink was obliterated by extending the anastomosis upward to include the kink, and after that the patient's progress was uneventful

She has been eating normally and gaining weight rapidly in the eight months since operation

CASE 9 —P C, a male child, was seen at the age of 6 months because of regurgitation of feedings He was found to have a sliding hernia with esophagitis and some stenosis Two attempts were made to reduce the hernia in the hope that the stenosis and ulceration would heal, but both were unsuccessful During the following four years, the patient required esophagoscopic dilatation six times

Operation (G H W ) was performed on Oct 25, 1955, as already described The intestine was reunited by side to side anastomosis This boy developed purulent sputum with some pulmonary collapse and a draining sinus in the chest wall, but a barium swallow, five months afterward, showed that all the anastomoses were working well He was taking a normal diet but his general development was still delayed by the chest infection

CASE 10 —B J, a man aged 31 years, had complained of dysphagia for fifteen years with notable deterioration in the four months previous to being seen Radiography and esophagoscopy demonstrated a short, narrow peptic stricture, 8 cm above the diaphragm, and a sliding hiatal hernia

Operation (G H W ) was performed on Nov 22, 1955 The steps were as described, the pyloromyotomy being done on the posterior surface Recovery and convalescence were uneventful and during the short time since operation the patient has been eating normal meals without discomfort and has been gaining weight

CASE 11 —F H, a boy of 16 years, had been under observation for vomiting and dysphagia since the age of 1½ years, and had had esophagoscopic dilatations six times with at the level of the projected gastric stoma. Intestinal continuity was restored by end-to-end anastomosis, and a gastrostomy performed. Pyloromyotomy was not done. Three days after operation the patient became distressed, and malodorous heavily bile-stained fluid was aspirated from his stomach. Radiographs showed a high fluid level in the stomach and another in the duodenum. The anterior end of the incision was opened and obstruction was found to be caused by acute kinking at the entero-anastomosis. Side-to-side anastomosis was made just below the kink and the spur of the kink divided. After this second operation, the patient was collapsed and dyspneic and was found to have a tension pneumothorax on the left side. This was aspirated and recovery thereafter was without incident.

For some short time he was rather afraid of taking solid food but he soon overcame this, and as his confidence increased he started to eat "all that was put before him" without discomfort. Strength and appetite improved rapidly until he had to diet against putting on too much weight. One of the most notable benefits of operation was the patient's ability to dine with friends. This altered his whole outlook on life.

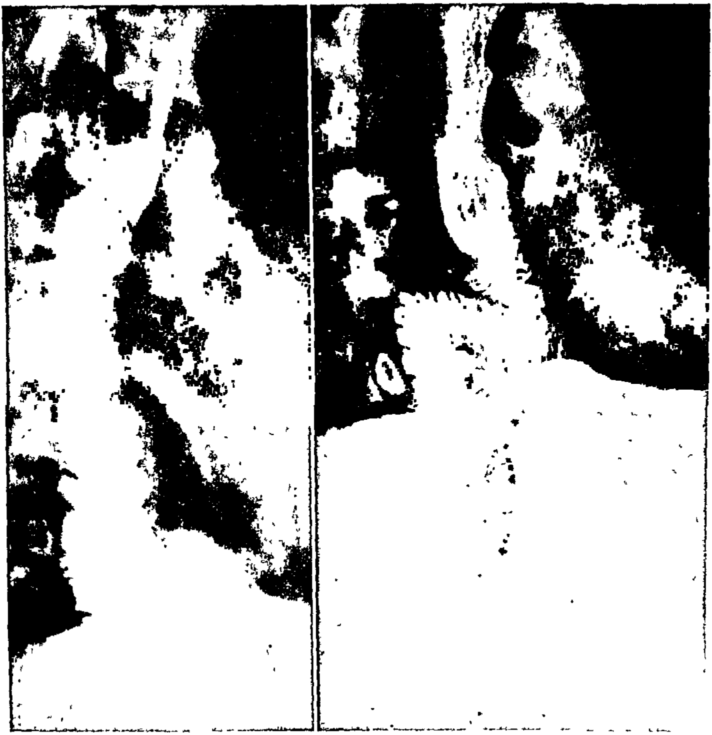
CASE 5.—E. B., a woman aged 47 years, was the only patient in this series for whom esophagojejunogastrostomy was done after the excision of a malignant lesion. The patient gave a long history of epigastric discomfort and belching for which she had taken alkalies. For six months she had had dysphagia and for a few weeks almost complete obstruction to solid food. She had lost 14 pounds weight. There were no abnormal physical signs. Radiography showed a stricture at the junction of the middle and lower thirds of the esophagus, and the top of this was seen, by esophagoscopy, to be at 32 cm. It was a squamous-cell carcinoma.

Operation (P. R. A.) was performed on Nov. 17, 1953. Some modification of the steps already described were adopted. A Roux loop was made long enough to reach the superior mediastinum. Instead of division of the vasa brevia, the upper part of the stomach down to the lower pole of the spleen on the greater curvature and halfway along the lesser curvature was removed with the spleen, body of the pancreas, and retroperitoneal tissues; the splenic and left gastric vessels being divided at their origin. This block of tissue with the fascia of the diaphragm was drawn up into the mediastinum and the esophagus dissected free, with its surrounding mediastinal tissue up to the aortic arch. Through the same skin and muscle incision, a further thoracotomy was made through the fifth space: two pairs of aortic intercostal arteries were divided and the arch of the aorta dissected free from the esophagus. The spleen was removed from the cardia so that the latter could be drawn up from beneath the arch of the aorta into the superior mediastinum and delivered from there into the pleural cavity. The Roux loop was threaded up through a hole in the transverse mesocolon, through the hiatus, beneath the aortic arch, and out into the left pleural cavity where it was stitched to the esophagus. The esophagus below, with the growth and the cardia, was removed and the esophagojejunostomy completed. The rest of the operation followed the lines laid down, eight arteriae rectae being divided, and the jejunal continuity restored by end-to-end anastomosis below the transverse mesocolon.

When assessed nineteen months later, the patient was eating four meals a day, had a good appetite, was maintaining her body weight and was free from symptoms. Dr. Johnstone reported: "barium swallow shows a satisfactory anastomosis at the level of the aortic arch. A little kinking of the jejunum is noted in its thoracic course but peristalsis is vigorous and effective. Barium passes through the hiatus and after a short course enters the stomach through a posterior stoma. Peristalsis of the stomach is only moderate."

CASE 6.—R. D., a boy who had vomited since birth, was first seen in the hospital at the age of 5 months. He was found at radiography and esophagoscopy to have an esophageal stenosis and sliding hiatal hernia. The stricture was dilated through the esophagoscope fifteen times during the next ten years. On frequent occasions, radiography demonstrated a large stomach with poor and ineffective peristalsis and pylorospasm, but at other times the stomach appeared normal.

as nearly equal as is possible to the length of the mesentery supplying it with blood vessels (Fig. 4, *A* and *B*). This is not always feasible, and in such case much can be done to improve a curled loop if the steps previously recommended are adopted (Allison, P. R. and Da Silva, L. T., 1953). If the hiatus is small it should be enlarged, but if the loop does not pass through it without kinking, it should be closed and a new hole made to suit the position of the intestine, for a sagging loop in the pleural cavity may cause stagnation or even obstruction; the jejunum should be bevelled to meet the back wall of the stomach without kinking; the lesser sac should be closed by stitching the stomach back to the diaphragm to prevent intraperitoneal herniation, that part



*A.* *B.*

Fig. 4.—*A*, A good Roux loop lying straight in the mediastinum After division of the vascular radicals the mesentery was as long as the gut

*B*, A less favorable Roux loop An arcade was deficient at one point so that vessels had to be divided far back toward the abdominal wall This left a curve that could not be straightened.

many long intervals of good progress. He had a short peptic stenosis with a large sliding hiatal hernia.

Operation (G. H. W.) was performed on Feb. 23, 1956. This was as described except that no pyloromyotomy was performed and intestinal continuity was achieved by end-to-end anastomosis. This junction became kinked and obstructed in the postoperative period and had to be converted into a large side-to-side anastomosis after which recovery was uneventful. A barium meal, one month after operation, showed uninterrupted progress of the medium through all segments of the gut and the patient is now eating well and is free from symptoms.

## DISCUSSION

Large esophageal defects created partly by disease and partly by surgical excision can be bridged by a length of jejunum, after a plastic operation on the mesentery according to the method of Roux, whose original idea of connecting the lower end of the loop to the stomach is applicable to reconstruction within the mediastinum, and would appear a priori to be the method least open to objection on metabolic grounds. The metabolism of the patients treated so far can be estimated by their appetite, their weight, their feeling of well-being and, in children, their development. On these criteria, the operation has fully justified itself and is superior to any other known method of reconstruction. Only by trial and waiting will it be learned whether gastrojejunal ulceration will prove to be an important complication. Although this did occur when the loop was used subcutaneously and was the reason why many surgeons abandoned the operation, the same will not necessarily occur when the mediastinal route is used, for here the point of entry of the jejunum into the stomach is at the top, in the region of the fundus, where the drainage is of mucus, and downward, rather than of acid pepsin, upward. The subcutaneous operation involves a loop of jejunum passing upward into the most dependent part of the stomach so that the jejunal mucosa must be constantly bathed by digestive juice. The occurrence of peptic digestion of the esophagus where this enters the top of the stomach in hiatal hernia is not comparable with the jejunum's entering the fundus of the stomach, for ulceration in the former circumstance is dependent upon the differential pressures in chest and abdomen which aspirate the juices into the esophagus, whereas in the operation of esophagojejunogastrostomy, the lower 10 to 15 cm. of the jejunum and its junction with the stomach are all under the same pressure in the abdomen. The loop is passed over the back of the fundus so that, as the stomach distends, it compresses the loop and prevents reflux. If the loop were to be brought straight through the hiatus and into the nearest part of the stomach, the chances of reflux acid ulceration would be greatly increased. This ''valve action'' might well be a menace if it were to prevent vomiting but, as already reported, one of the patients has vomited successfully. It would be interesting to know how he managed this.

The main objection to the operation is the tediousness of the techniques and the consequent opportunities for mistakes. From the errors so far committed, some advice can be tentatively offered; the Roux loop should be as straight as may be, which means that the length of the intestine should be

# INCOMPETENCE OF THE CARDIA

Nils Stensrud, M D
Oslo, Norway

REFLUX of gastric juice in the esophagus causes troublesome symptoms Especially after meals, the patients have a feeling of discomfort in the epigastrium or behind the lower part of the sternum They complain of heartburn and occasionally, sour gastric content may come up into the mouth In cases of pronounced esophagitis, the patients have a burning pain behind the sternum which is more intense after meals It is characteristic that the pains are felt in certain positions, when the patients lie flat on their backs or when they bend forward, positions which facilitate regurgitation

The mucous lining of the esophagus exhibits pathologic alterations, from a slight inflammatory condition to ulcerations with fibrosis and strictures These alterations are most commonly located in the lower part of the esophagus

Hiatus hernia is the most frequent cause of reflux of gastric juice The cardiac end of the stomach slides up into the thoracic cavity, and then the cardia lies above the diaphragm with the result that free regurgitation of gastric juice occurs (Fig 1)

In rare cases, there may be typical regurgitation symptoms and the same pathologic alterations in the esophagus, although no hiatus hernia can be demonstrated radiologically

In such cases there is an incompetent cardia It is believed that the cause is due chiefly to an abnormal conjunction of the esophagus with the stomach, as shown in Fig 2 The normal esophagogastric angle is here straightened out so that the esophagus empties into the stomach at its highest point Allison[1] has shown that when the stomach and esophagus are taken from a body and placed in normal relation to each other, water which is pumped into the pylorus will stop when it reaches the cardiac If the parts are placed in such a manner that the esophagus empties into the top of the stomach, the cardia will be insufficient

In normal dogs, the gastric contents cannot be emptied into the esophagus as long as they lie in normal relation to each other, but if the fundus is removed, regurgitation will occur

Barret[2] demonstrated regurgitation in normal individuals when he depressed the fundus during operation, and it is believed that it is depression of

---

From Surgical University Clinic A Rikshospitalet Head Leif Efskind M D Professor of Surgery

Received for publication June 11 1956

of the loop below the gastric anastomosis may need to be trimmed to bring it down to lie snugly on the patients' right side of the duodenojejunal flexure; this part of the loop should be joined by a large side-to-side anastomosis to those few inches of jejunum still in continuity with the duodenum, for an end-to-end junction is too liable to become obstructed by slight rotation, kinking, or adhesion formation; the patency of this junction is vital for the other anastomoses. Pyloromyotomy is advised so that the weak peristaltic waves of the stomach are not obstructed by a pylorus in spasm from vagotomy. A temporary gastrostomy is of great value for letting out the quantities of air that the patient swallows. The gastrostomy tube should be left in for at least ten days until a safe track of granulations has formed, but if the stomach is emptying freely and aerophagy has ceased it may remain plugged for the last five days before removal. If for any reason the abdomen has to be reopened soon after operation, great care is needed to avoid inhalation of intestinal contents; a radiograph of the chest should be taken before the patient returns to the ward, for air entering the abdomen may be aspirated into the pleura during operation, or forced up when the abdomen is closed, and so cause severe respiratory embarrassment.

## SUMMARY

1. Esophageal replacement by an isolated loop of jejunum in the mediastinum is described.

2. The technique of esophagojejunogastrostomy and its metabolic advantages are discussed. It is considered to be particularly applicable to the treatment of esophageal stenosis in children and in women, who do not tolerate exclusion of the stomach as well as men do. Where the more complicated operation is not contraindicated, however, it may become the routine operation for all patients with long, benign stenoses.

3. Combined with adequate lymphatic dissection, the operation may be applicable to patients who have carcinoma of the esophagus not involving the stomach.

4. Brief case histories are given of 11 patients treated in this way, with one death. There were many complications due to technical faults, and these are described. After the complications had been overcome, the health of the 10 living patients remains excellent.

## REFERENCES

1. Brain, R. H. F.: Steatorrhoea in Oesophago-Gastric Surgical Practise, Proc. Royal Soc. Med. 46: 438-444, 1953.
2. Roux, C.: Sem. médica, Paris 27: 37, 1907.
3. Allison, P. R., and Da Silva, L. T.: The Roux Loop, Brit. J. Surg. 41: 173-180, 1953.

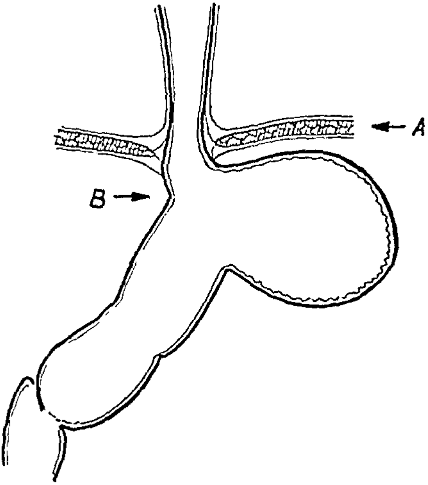


Fig. 2.—*A*, Diaphragm. *B*, Abnormal right-angled conjunction of the esophagus with the stomach.

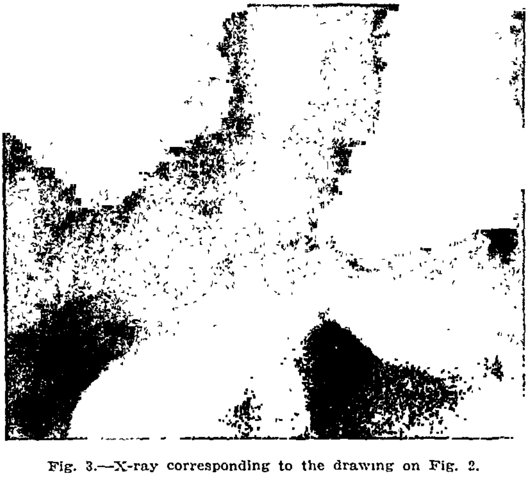


Fig. 3.—X-ray corresponding to the drawing on Fig. 2.

the diaphragm with the fundus, which facilitates vomiting in human beings with normal cardia function.

At the Surgical Dept. A. Rikshospitalet, a case of cardia incompetence has been treated.

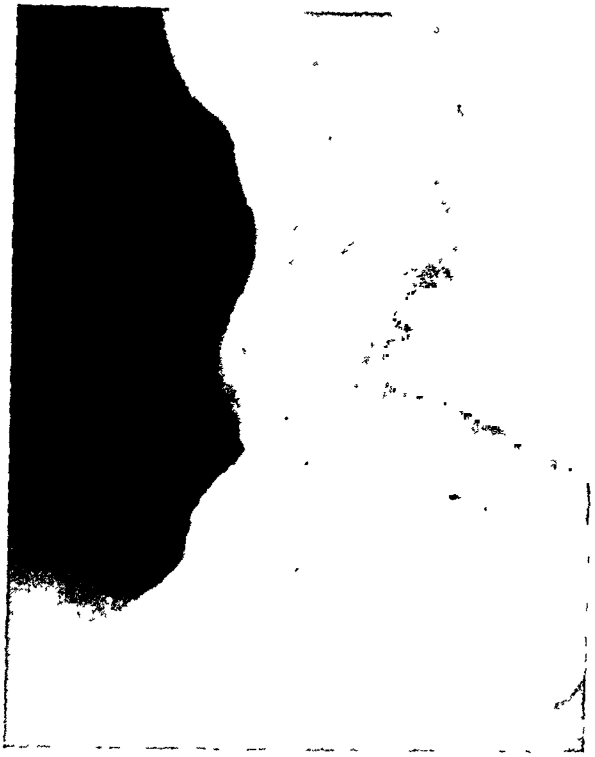


Fig. 1.—X-ray of sliding hernia.

## CASE REPORT

*Case history:* The patient was a 47-year-old man who had had gnawing pains behind the lower part of the sternum, radiating down into the stomach for twenty years, and he had also suffered from regurgitation. He had three times previously, in 1935, 1947, and 1953, been taken into hospital for observation for these symptoms, but examination including x-ray of the stomach revealed no explanation. In addition to these symptoms, he had had difficulty in swallowing during the past few years and had the feeling that food stopped behind the lower part of the sternum. He was admitted to the department under the diagnosis of tumor of the esophagus, as x-ray examination had revealed an irregular contour in the lower part of the esophagus.

*Present condition:* He was in good general health. General examination revealed nothing pathologic. Blood was normal. Feces, benzidine five seconds. The Ewald test demonstrated acidity to be 15/40. X-ray of esophagus, stomach, and duodenum showed an irregular contour in the lower part of the esophagus with a small niche, but no tumor infiltration. There was marked regurgitation of the stomach contents. The stomach had an atypical position, lying horizontally, with the greater part of it in the right side of the abdomen. The junction of the esophagus with the stomach was pathologic, as shown in Figs. 2 and 3. The radiologists' conclusion was esophagitis and insufficiency of the cardia.

Esophagoscopy revealed an inflamed thick mucous membrane in the lower part of the esophagus and severe regurgitation, but no signs of ulceration.

An operation was performed through a thoraco abdominal incision. The hiatus was normal. The junction of the esophagus with the stomach was found to be a right angle, anteriorly. The fundus was then sutured upward laterally and to the left of the esophagus It was fastened to the left dome of the diaphragm and farther, with interrupted sutures, downward along the esophagus (Fig. 4).

There were no postoperative complications. Control examination six months after the operation found the patient free of symptoms. He was completely satisfied with the result of the operation X-ray of the esophagus, stomach, and duodenum now shows a normal relation between the esophagus and stomach (Fig 5) and there is no regurgitation Esophagoscopy revealed a slight esophagitis, considerably less than at the postoperative examination, also without regurgitation.

## DISCUSSION

I have found no reports in the literature of similar cases in which an operation has been performed. But Barret[2] mentions that Lortat-Jacob has performed several operations on similar cases with good result

We believe that this condition, an incompetent cardia without hiatus hernia, occurs but rarely. This condition is not always diagnosed. It seems that these patients can be cured by a minor operation

## SUMMARY

A case of cardiac insufficiency without hiatus hernia was treated surgically by a plastic operation with a good result.

## REFERENCES

1 Allison, P R Reflux Esophagitis, Sliding Hernia and the Anatomy of Repair, Surg, Gynec & Obst 92: 419, 1951.
2 Barret, N R Hiatus Hernia, Brit J Surg 42: 231, 1954.
3 Stensrud, N: Hiatus Hernia, Acta chir. scandinav. 107: 57, 1954.

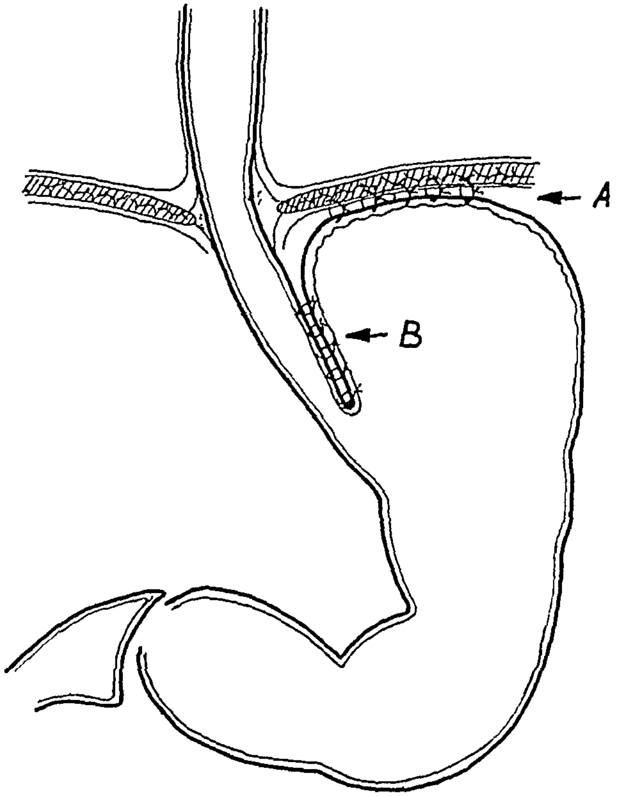


Fig 4 —*A*, Sutures between diaphragm and fundus of stomach. *B*, Sutures between esophagus and stomach restoring a normal esophagogastric junction

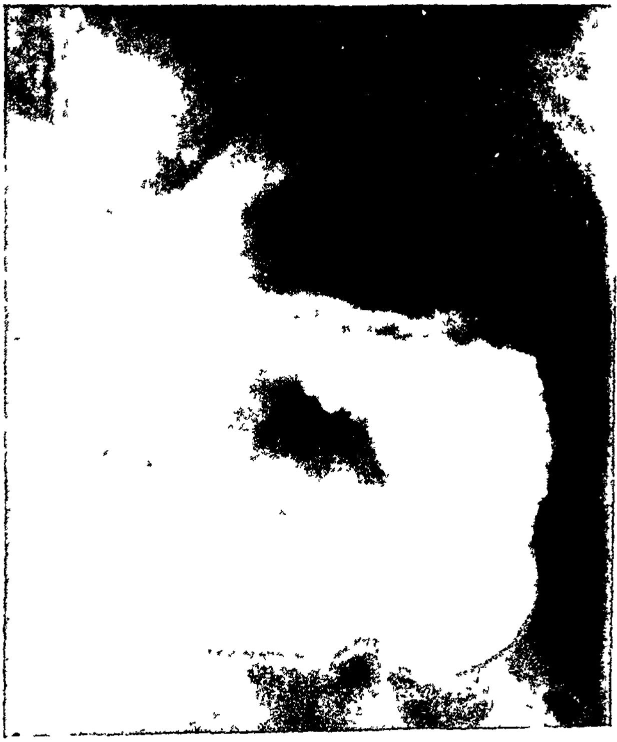
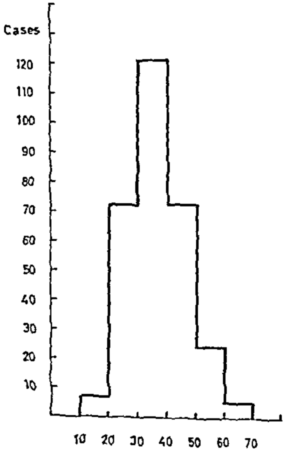


Fig. 5 —Postoperative x-ray corresponding to Fig. 4.

in Table I. Twenty-seven patients had an open cavity in the contralateral lung. In more than one-half of the material, 54.5 per cent (164 cases), it was necessary to do an extrapleural resection or a decortication.

## AGE

Old age is not in itself a contraindication to the removal of a lobe with cavitary tuberculosis. Due to emphysema and fibrosis of the remaining lung, it will often be found necessary to add an osteoplastic thoracoplasty at the same operation. There were 5 patients in this material operated upon at an age above 60 years. In all 5 cases, a lobectomy with simultaneous osteoplastic thoracoplasty was carried out. Four patients withstood the procedure very well. The fifth patient, a 64-year-old man died from circulatory insufficiency two days after the operation.



[illegible]

# LOBECTOMY FOR PULMONARY TUBERCULOSIS

## An Analysis of 301 Cases

Viking Olov Björk, M.D.
Stockholm, Sweden

LOBECTOMY for pulmonary tuberculosis was already introduced in 1945 by Overholt and Chamberlain. The results have improved due to the antimicrobial therapy. The aim of this paper is to report a group of 301 cases. One-half of this group has been followed for one to five years. The immediate and late results are given with special reference to the lung function after lobectomies with different space-diminishing procedures.

### MATERIAL

In this group, even patients with a very bad pulmonary function have been accepted. Of the 301 patients, 10 are dead and 163 have been followed. Ninety-one patients were excluded as the observation time was less than one year. Twenty-three patients have refused follow-up tests mainly because they did not like to have another bronchospirometry. Fourteen patients have not been traced.

Only patients with lobectomies are included, except in 39 cases of the 301 in which a wedge resection was added to the lobectomy. All patients with segmental resections are reported later.

Nearly one-half of the patients, i.e., 48.5 per cent, had bilateral pulmonary tuberculosis. The extent of the tuberculosis in the contralateral lung is shown

Table I. The Condition of the Contralateral Lung at the Time of Operation

| CONDITION OF THE CONTRALATERAL LUNG | NUMBER OF CASES |
|---|---|
| Normal | 155 |
| Old pleurisy | 5 |
| Small infiltration | 69 |
| Infiltration earlier treated by pneumothorax | 19 |
| Infiltration being treated by pneumothorax | 6 |
| Cavity "closed" by conservative treatment including chemotherapy | 5 |
| Cavity "closed" by pneumothorax | 10 |
| Cavity "closed" by thoracoplasty | 4 |
| Open cavity | 17 |
| Open cavity under pneumothorax | 7 |
| Open cavity under thoracoplasty | 3 |
| Pneumonectomy | 1 |
| Total | 301 |

---

From Sabbatsberg Hospital; Surgeon in Chief: C. Crafoord, M.D., Professor. St. Göran Hospital; Physician in Chief: A. Westergren, M.D., Professor, and Söderby Hospital; Physician in Chief: T. Bruce, M.D., Docent.

This work was aided by The Swedish National Association Against Tuberculosis.

Received for publication May 29, 1956.

*6. Secondary Infected Congenital Cyst.*—One patient was followed with the diagnosis of congenital cyst from 8 years of age when a Mantoux test was negative. Later he developed a tuberculous infection in the congenital cyst and, at the age of 22, a resection of the right lower and middle lobes was performed.

### PREOPERATIVE INVESTIGATION OF LUNG FUNCTION

The most important information of the lung function was obtained by a study of all x-ray films from the beginning of the disease. The maximal movements of each diaphragm are also recorded.

*1. The Maximal Breathing Capacity (MBC).*—The lowest preoperative value for MBC was 20 L. per minute in a 31-year-old woman who had had bilateral pneumothorax treatment. Before the operation she had had a long period of breathing exercise training and increased the MBC to 32 L. per minute. After resection of the right upper and middle lobes under an old 3-rib thoracoplasty the patient awoke but failed to get strength enough to take over adequate spontaneous ventilation and died. This happened before we introduced the prolonged postoperative artificial ventilation. The lowest preoperative value for a surviving patient was 26 L. per minute in a 50-year-old woman who had a resection of the right upper lobe and a wedge resection of the lower lobe under an old 5-rib thoracoplasty. This patient is still doing full time office work five years after operation. The MBC was below 69 L. per minute in 50 per cent of the women and below 49 L. in 14 women.

In 50 per cent of the men, the MBC was below 100 L. per minute and 9 cases had an MBC of less than 49 L. per minute. The lowest preoperative MBC in a man was 28 L. per minute. This patient was 33 years old and had had a left 7-rib thoracoplasty and then a right upper lobectomy under a 3-rib thoracoplasty. He survived the operation with spontaneous ventilation and, two years later, is working full time as an engineer and his MBC is still 28 L. per minute.

*2. The Vital Capacity.*—The lowest preoperative value for vital capacity was 0.84 L. in a 42-year-old woman with a left-sided 8-rib thoracoplasty fifteen years old. Eighty-nine per cent of the total oxygen uptake came on the right side where the upper and middle lobes and a wedge of the lower lobe were resected and an osteoplastic thoracoplasty performed at the same time. The patient withstood the operation well with the aid of prolonged artificial ventilation but died fourteen months later from cor pulmonale.

*3. Bronchospirometry.*—One of the most valuable preoperative investigations was bronchospirometry. Due to the instrument designed by Carlens, it has been possible to obtain a number of postoperative determinations and these findings are reported under "Results."

*4. The Residual Capacity.*—The residual capacity was investigated in 153 cases. It was found to be below 35 per cent of the total capacity in 130 cases, between 35 and 40 per cent in 16 cases, between 40 and 45 per cent in 5 cases, and more than 45 per cent in 2 cases.

*5. The Pulmonary Artery Pressure.*—The pressure in the pulmonary artery was only recorded in 15 cases and, in most of these patients, during cr

TABLE II. *AGE AND SEX DISTRIBUTION IN 301 PATIENTS UNDERGOING LOBECTOMY FOR PULMONARY TUBERCULOSIS*

| AGE | MEN | WOMEN | TOTAL |
|---|---|---|---|
| 10-19 | 3 | 4 | 7 |
| 20-29 | 37 | 35 | 72 |
| 30-39 | 61 | 60 | 121 |
| 40-49 | 46 | 26 | 72 |
| 50-59 | 19 | 5 | 24 |
| 60-69 | 4 | 1 | 5 |
| Totals | 170 | 131 | 301 |

## INDICATIONS FOR LOBECTOMY

*1. Tuberculous Cavitation.*—The most common indication for the lobectomy has been cavitary tuberculosis in 85 per cent, or 255 cases. The right upper lobe was the most common localization, i.e., 59 per cent compared to 26 per cent left upper lobectomies for cavitation. This very high incidence of right upper lobectomies will be explained by the fact that on the left side a segmental resection of the apicoposterior and eventually also the anterior segments is very often carried out for cavitary tuberculosis. Although I have always tried to leave the anterior segment of the right upper lobe when it contained no palpable tuberculosis there was actually a high frequency of lobectomies.

In comparatively few cases, a lower lobe cavity was the indication for a resection, i.e., 28 cases, or 11 per cent, on the right side, and 11 cases, or 4 per cent, on the left side.

There was no case of isolated cavitary tuberculosis in the middle lobe. However, in 22 cases, or 8 per cent, the middle lobe was resected for cavitary tuberculosis, in 14 cases, or 5 per cent, in combination with the upper lobe, and in 8 cases, or 3 per cent, in combination with the lower lobe.

*2. Bronchial Stenosis.*—Bronchial stenosis was the indication for resection in 26 patients, i.e., 9 per cent of the material. This stenosis was localized to the right main bronchus in 2 cases, to the left main bronchus in 3 cases, and in all 5 patients the upper lobe bronchus was also stenosed. The lobar bronchus alone was involved in the right upper lobe in 11 cases, in the right middle lobe in 2 cases, the right middle and lower lobes in 4 cases, left upper lobe in one case, and in the left lower lobe in 3 cases.

*3. Bronchiectasis.*—Bronchiectasis with positive sputum was the indication for resection in 6 patients, or 2 per cent (2 right upper, 1 left upper, and 3 in the right middle and lower lobes). All these patients had had bronchial disease and showed some narrowing or deformation of the lobar bronchus.

*4. Tuberculous Pneumonia.*—Tuberculous pneumonia was the indication for resection in 6 cases, or 2 per cent. The right upper lobe was involved in 4 cases, the right lower lobe in one case, and the left upper lobe in one case.

*5. Tuberculous Empyema With Bronchial Fistula.*—In 7 cases, a tuberculous empyema with bronchial fistula was the indication for resection. Two of these fistulas were caused by an extrapleural plomb of plastic material (Polystan).

grown-up patients. I have therefore lately mobilized the diaphragm around its periphery and resutured it to the fifth intercostal muscle. Three such cases are included in this material.

The lobectomy was performed under an old thoracoplasty of 7 ribs in 19 cases, of 5 ribs in 26 cases, and of 10 ribs in one case. This thoracoplasty had been performed one to fourteen years before the resection.

In 3 cases a preliminary plomb had caused perforation into a tuberculous cavity with empyema. The plomb was removed in one piece with the chest wall and the lobe. The chest wall was resected together with an empyema and a lobe after an extrapleural pneumothorax in one case.

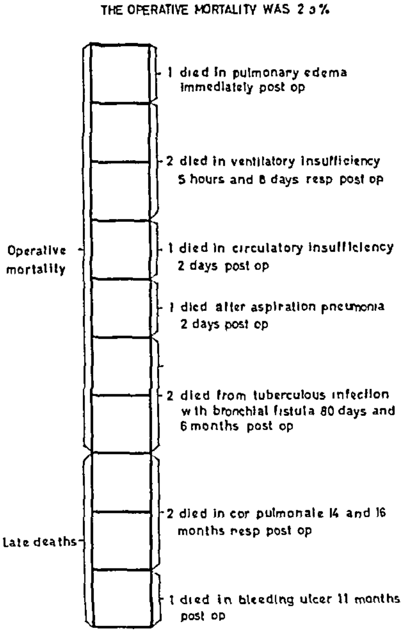

THE OPERATIVE MORTALITY WAS 2.3 %
1 died in pulmonary edema immediately post op
2 died in ventilatory insufficiency 5 hours and 8 days resp post op
Operative mortality
1 died in circulatory insufficiency 2 days post op
1 died after aspiration pneumonia 2 days post op
2 died from tuberculous infection with bronchial fistula 80 days and 6 months post op
2 died in cor pulmonale 14 and 16 months resp post op
Late deaths
1 died in bleeding ulcer 11 months post op

Fig. 2.—The operative mortality was 2.3 per cent or 7 cases. There were 3 late deaths.

## RESULTS

*Dead.*—Altogether, 10 patients are dead.

*The operative mortality* was 2.3 per cent (7 cases).

One 40 year old woman had a right upper lobectomy under an old 5 rib thoracoplasty. When the wound was closed and immediately after injection of Prostigmin she suddenly developed pulmonary edema. Clear fluid poured out from both lungs in such an amount that it could not be effectively [illegible] away through the openings of a Carlen's double [illegible] catheter. As I [illegible]

The highest value was 35 mm. Hg mean pressure in a 50-year-old woman. She is, five years after operation, doing office work full time. On the other hand, the mean pressure in the pulmonary artery during operation in a 42-year-old woman was only 23 mm. Hg and yet she died from cor pulmonale fourteen months afterward. From this limited experience, it seems difficult to make any long time prognosis based on the pulmonary artery pressure. The lowest value was 7 mm. Hg mean pressure in a patient with a pulmonary valvular stenosis, where the right ventricular pressure was 171/4 mm. Hg. Pulmonary resection and a valvulotomy through the pulmonary artery was successfully performed and the right ventricular pressure returned to 24/3 mm. Hg and the pulmonary artery pressure rose to 17/6 mm. Hg (mean pressure 12 mm. Hg).

## METHOD OF TREATMENT

In only 18 cases (6 per cent) was the lobectomy performed without a *space-diminishing procedure.* In only 2 patients in which a thoracoplasty was omitted at the time of resection was it necessary to add it after 11 days and 5 months later, respectively, to obliterate a remaining space.

When the decision was made to add a space-diminishing procedure to the resection, the quantity of the resected lung as well as the quality of the remaining lung parenchyma had to be considered. I have usually added a space-diminishing procedure under the following circumstances: (1) when more than one lobe is resected, (2) palpable tuberculosis in the remaining lung, (3) contralateral disease, (4) after resection of a tuberculous empyema, (5) after an accidental opening of a tuberculous cavity or an empyema, and (6) when the remaining lung is emphysematous and fibrotic and does not fill the space.

A one-stage procedure is always to be preferred. In the beginning, a preliminary 3-rib (33 cases, i.e., 11 per cent), or a 5-rib thoracoplasty (14 cases, i.e., 5 per cent) was performed and after a few months up to a year later the resection was performed with additional rib resection if considered necessary.

Later a one-stage operation has always been used. First, an extrapleural Polystan plombage was used in 10 cases, then a tailoring thoracoplasty with resection of posterior parts of ribs 2 to 5, 6, or 7 was performed.

To avoid the immediate danger of paradoxical movements of the chest wall with carbon dioxide retention and the late hazards of infection in a foreign body, I designed an osteoplastic thoracoplasty with fixation of the posterior parts of the ribs by nylon sutures through drill holes. The first rib is usually left behind. The second rib is incised close to the sternum and placed as close as possible to the mediastinum, its posterior end being fixed to the costovertebral ligaments by a thread behind the posterior tip of the intact sixth rib. Then a row of five to ten isolated sutures will approximate the first intercostal muscle bundle (attached to the second rib) to the mediastinal pleura. The addition of an osteoplastic thoracoplasty to a lobectomy has been performed in 149 cases in this material.

Although the cosmetic result has been very good, with the main deformity in the axilla, and the arm movements practically unimpaired, I like to avoid thoracoplasties in young growing individuals and when possible even in the course In 2 other cases of segmental resection where a hemothorax complicated the postoperative course, re operation was therefore performed after twelve and thirty six hours, respectively, and then the recovery was uneventful I will therefore continue to re operate on patients with a postoperative hemothorax as early as possible to avoid the development of an infected hematoma and a bronchial fistula

*Infection* · In one case, an extrapleural Polystan plomb had been introduced after a right upper lobectomy It became infected and caused a bronchial fistula necessitating extirpation of the plomb and the chest wall corresponding to the upper 8 ribs in two stages before healing occurred

A tuberculous cavity was opened on four occasions and resulted in a tuberculous empyema with prolonged drainage treatment needed in 2 cases

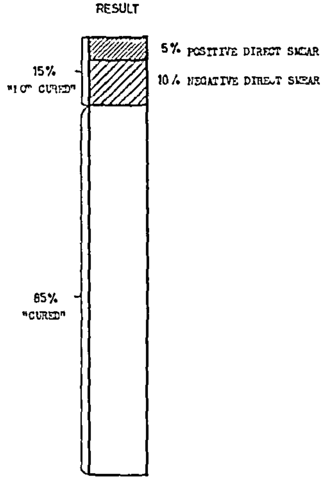


Fig 3 —Eighty five per cent were considered cured Only 5 per cent were positive on direct smear

*Suture granulomas* in the bronchus were found in 14 5 per cent of the cases when stainless steel or silk sutures were used, and had to be removed through the bronchoscope before I switched over to 30 day chromic catgut (Ethicon)

*"Cured Patients"* —

As the aim of the surgical treatment of pulmonary tuberculosis is to make the patient free from tuberculous bacilli in the sputum the following criteria of a "cure" were chosen (1) negative guinea pig test on gastric washing and sputum negative culture of sputum, and negative direct smear on three consecutive days, and (2) no active disease found on x ray examination All these criteria were fulfilled in 125 of the "cured" patients

In the remaining 14 cases it was not possible to obtain sputum and therefore only the gastric washing test was performed and found negative Altogether a "cure" was obtained in 139 cases of 163 followed up, i e , 85 per never had such an experience before, I did not remove the Carlen's tube and introduce a bronchoscope for a more effective aspiration, which might have saved the patient. As a consequence, I now do not use the Carlen's double lumen catheter for anesthesia except in a few very wet cases, but have adopted the Overholt prone position for most patients.

Two patients died from ventilatory insufficiency five hours and eight days after the operations, respectively. Both patients had had bilateral pneumothorax earlier and were operated upon before we introduced the prolonged postoperative artificial ventilation.

One 64-year-old man died two days after operation from circulatory insufficiency.

One 34-year-old woman had had a left pneumonectomy three months earlier. At a second operation, a right upper lobectomy was performed as well as an osteoplastic thoracoplasty. She was in good condition for two days when she vomited, developed aspiration pneumonia, and died. She, too, was operated on before the introduction of prolonged artificial ventilation. With this treatment, an aspiration is prevented due to the cuffed tracheal cannula.

Two patients, both having tuberculous empyema before operation, died from the tuberculous infection opening the bronchus, one within eighty days, the other six months after operation. There were three late deaths. Two patients died fourteen and sixteen months, respectively, after operation from cor pulmonale. The third patient died eleven months after operation from a bleeding ulcer.

*Other Complications.—*

*Spillover* was encountered in 2 cases. In one of these, there was a contralateral pneumothorax and a tracheostomy had to be performed. Patients with a pneumothorax on the contralateral side or with fixation of the diaphragm due to an old pneumothorax usually have difficulty in producing an effective cough and therefore a tracheostomy has to be added for aspiration of secretions and for diminishing the dead space as the respiratory work is greatly increased in these cases.

*Ventilatory insufficiency* was the reason for a tracheostomy in 3 cases, and the addition of postoperative prolonged artificial ventilation in 5 cases.

*A residual space* necessitated the insertion of a drainage tube in 4 patients and an additional thoracoplasty eleven days later in one case, and five months after operation in another case.

*Hemorrhage:* A severe postoperative hemothorax was encountered in 4 cases. The hematoma was not evacuated in 2 patients resulting in infection which necessitated an extrapleural pneumonectomy in one case and resection of the middle lobe and the chest wall in another case. The hematoma was evacuated after seventeen days in one case, but first a staphylococcus then a fungus infection necessitated a 3-month period of drainage with local chemotherapy.

In the fourth case, the hematoma was evacuated within twenty-four hours after operation and this patient then had an uncomplicated postoperative operated side straight up in the air (180 degrees) One patient could bring this arm to 175 degrees, 8 patients to 170 degrees 6 patients to 165 degrees, one patient to 160 degrees, 3 patients to 150 degrees, 3 patients to 140 degrees, one patient to 120 degrees, and one patient to 90 degrees

Although this group includes 3 patients with chest wall extirpation in one block with infected plombes who could only bring up the arm on the operated side to 160, 150, and 90 degrees, respectively, there were many more patients with impaired arm movements than in the group with osteoplastic thoracoplasty This difference is statistically significant ($0.01 > P > 0.001$)

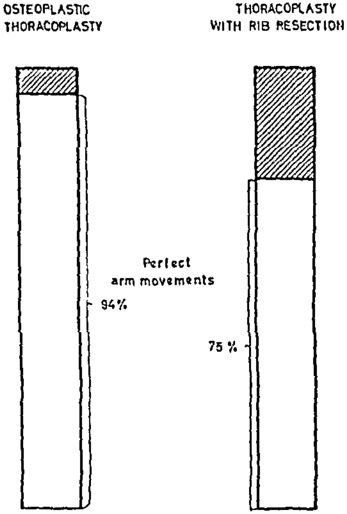


Fig [illegible] —There was a considerably better cosmetic and functional result regarding the arm movements in the patients having an additional osteoplastic thoracoplasty as compared to patients having thoracoplasty with rib resection This difference is significant

One explanation of the better functional result when an osteoplastic thoracoplasty was added to the resection is that this was a one stage procedure, while, in the other group, thoracoplasty with rib resection, at often two and sometime several stages, was performed Naturally a division of the periscapular muscles several times will give a bad functional result

*Pain* During the first months after an osteoplastic thoracoplasty, several patients experienced some pain on the operated side, sometimes radiating out into the arm There was also a loss of sensitivity because the upper 5 intercostal nerves are divided Leaving the first rib has, in one patient, caused radiating pain in the arm, necessitating the removal of the rib After one year, 80 per cent of the patients with osteoplastic thoracoplasty were completely free from pain The remaining 10 patients complained about the following discomfort cent. A negative direct smear was found in 17 of the 24 patients considered "not cured," making a total of 156 cases, i.e., 96 per cent with a negative direct smear.

Two of the "cured" patients had had some reactivation shortly after the operation but reacted promptly to antimicrobial therapy.

*Working capacity:* Of the "cured" 139 patients, 110, or 79 per cent, were working full time one year after operation. Twenty-six different professions were represented. Housework and office work were most frequently found, although some patients performed more heavy work. Twelve patients performed a one-half day's work and 17 did not work at all. Of these nonworking patients, 2 had been in sanatoriums for eight and thirteen years, respectively, and had never performed any work. One patient was not allowed to take up work by her mother. Five patients had done heavy work before surgery and could not resume the same work.

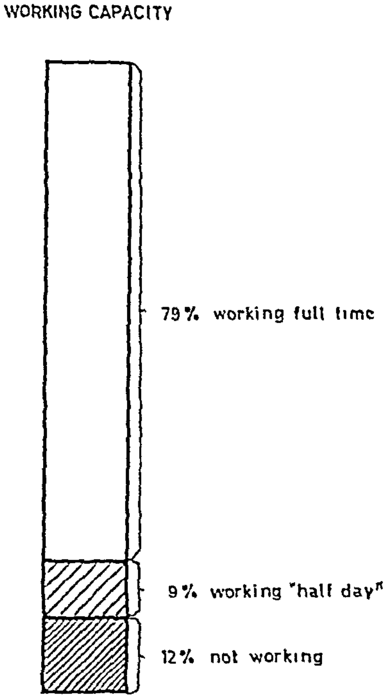


Fig. 4.—The working capacity.

*The arm movements:* The postoperative arm movements were recorded and compared in 2 groups of patients having had thoracoplasties.

1. In 50 cases with an osteoplastic thoracoplasty, 47 cases, or 94 per cent, had perfect arm movements. In 3 cases, there was a deficient ability to bring the arm on the operated side straight up in the air (i.e., 180 degrees). Two patients could bring the arm to 175 degrees and one patient having had a tuberculous empyema in the apicolysis space could only bring the arm to 160 degrees.

2. In 93 patients having had conventional thoracoplasties with rib resections, the arm movements were normal in 70 cases, i.e., 75 per cent. Twenty-three cases, or 25 per cent, had a deficient ability to bring the arm on the

*Cough:* Seventy per cent of the patients did not have any cough. The remaining 30 per cent experienced some irritating cough or had some expectoration in the morning. Patients with an irritating cough were bronchoscoped and, in 17 cases or 35 per cent of these patients, sutures were removed from the bronchial tree. Several patients had coughed up sutures. But silk or stainless steel sutures were removed from the bronchial stump as long as four years after operation. Some of these patients were positive to guinea pig test, but turned negative after removal of sutures and suture granulomas containing tuberculous tissue. I have therefore adopted the use of 30 day chromic catgut on Atraumatic needles (Ethicon) for bronchial closure and bronchotracheal anastomosis.

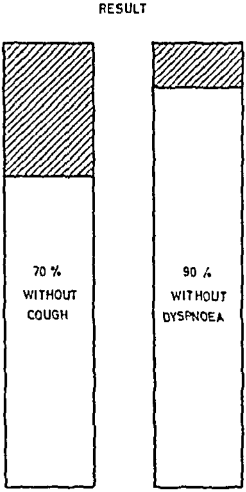


Fig. 7.—As many as 30 per cent of the patients had cough at the follow up. Only 10 per cent complained of shortness of breath.

*Dyspnea:* Ninety per cent of the patients did not feel short of breath. The remaining 10 per cent complained of dyspnea. In the patients with a subjective feeling of shortness of breath, the maximal breathing capacity varied from 76 to 16 L. per minute for the male and from 64 to 22 L. per minute for the female patients.

*Not "Cured" Patients.—*

*Late reactivation:* Six patients with bilateral disease at the time of operation had reactivation, in both lungs, one to two years after resection in 4 cases, and cavitation in the remaining lung after operation in 2 cases. Five patients were readmitted for antimicrobial therapy, and one for a new resection.

*Contralateral disease:* In 8 cases, the disease in the contralateral lung was considered the origin of the tuberculous bacilli. In 5 of these

Four patients had some pain radiating out into the arm. (The pain was not intensive enough to warrant the removal of the first rib.) Two patients complained of some pain under the scapula and in one of them a bursa was removed under the scapula. In 2 other cases, a bursa was found but disappeared on repeated aspirations with injection of dihydrocortisone.

One patient had pain in the side of the thoracoplasty when there was a change in the weather. One patient noticed some discomfort in the third costochondral junction and 2 patients had some pain when the scapula during maximal movements jumped over the sixth and seventh ribs, respectively.

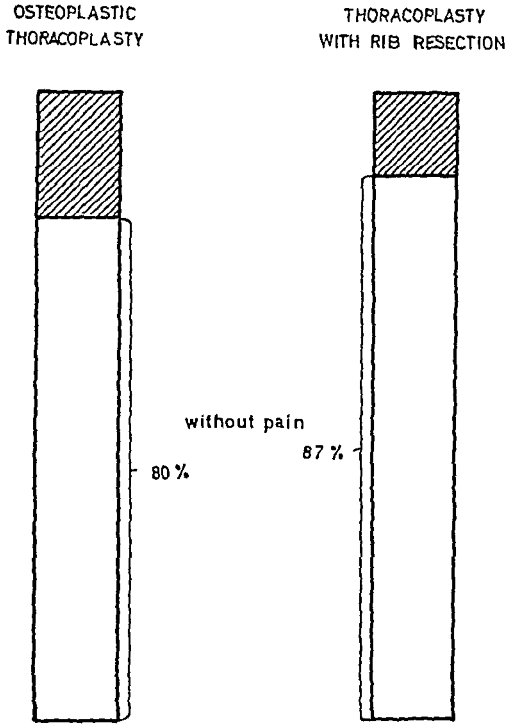


Fig. 6.—The incidence of pain was slightly higher in the patients having an osteoplastic thoracoplasty. This difference is not significant.

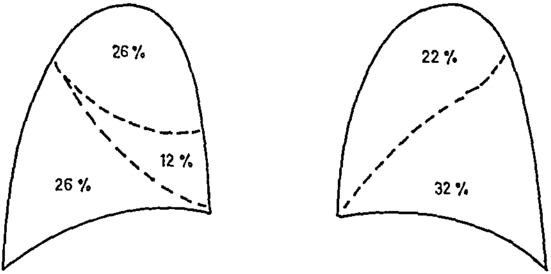
In the second group of patients with rib-resection thoracoplasties, 81 cases, or 87 per cent, were free from pain. Eleven patients complained of some pain: 2, in the arm; 4, under the scapula; 3, when the scapula jumped over the intact seventh rib; and, in one case when scapula jumped over the intact sixth rib. One patient complained of pain over the lower costal margin. Furthermore, the scapula jumped over or could fasten under the seventh rib or some rib regeneration in 6 cases without causing the patient any pain.

Somewhat more pain is therefore found postoperatively in the group with osteoplastic thoracoplasty although the difference is not great and is not statistically significant ($P = 0.20$). The cosmetic and functional result with the osteoplastic thoracoplasty, however, is far superior. The group with rib resection has been followed longer than the group having had an osteoplastic thoracoplasty. As the pain usually disappears with time, especially after the patient is fully occupied with his work, the five-year follow-up may be more favorable.

TABLE III THE POSTOPERATIVE FUNCTIONAL RESULT IN UNILATERAL CASES OF PULMONARY TUBERCULOSIS

| OPERATION | | NUMBER OF CASES | FUNCTION OF THE OPERATED SIDE IN PER CENT OF THE TOTAL | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | OXYGEN UPTAKE | | VENTILATION | | VITAL CAPACITY | |
| | | | MEAN VALUE | RANGE | MEAN VALUE | RANGE | MEAN VALUE | RANGE |
| Resection right upper lobe | with osteoplastic thoracoplasty | 15 | 38 | 27 49 | 50 | 37 69 | 38 | 34 48 |
| | resection costae 1 5 in 2 stages | 7 | 41 | 32 49 | 42 | 36 49 | 39 | 28 42 |
| | +costae 1 7 in 2 3 stages | 4 | 38 | 29 39 | 47 | 36 59 | 46 | 33 63 |
| | +costae 1 8 in 3 4 stages | 2 | 38 | 30 46 | 39 | 35 42 | 32 | 27 36 |
| Resection right upper and middle lobes | +costae 1 7 | 3 | 26 | 23 28 | 39 | 31 41 | 32 | 22 40 |
| Resection right middle lobe | | 1 | 42 | | 46 | | 47 | |
| Resection right lower lobe | +apical plombage | 1 | 21 | | 39 | | 22 | |
| | +costae 7 11 | 1 | 36 | | 13 | | 39 | |
| Resection right lower and middle lobes | without thoracoplasty | 1 | 26 | | 49 | | 36 | |
| | +costae 1 6 or 1 8 | 2 | 26 | 25 26 | 34 | 38 49 | 33 | 29 35 |
| | +costae 4 10 or 7 10 | 2 | 23 | 23 23 | 33 | 32 33 | 25 | 24 26 |
| | +apical extrapleural plombage | 1 | 16 | | 29 | | 24 | |
| | +osteoplastic thoracoplasty | 2 | 25 | 24 25 | 36 | 34 38 | 32 | 27 36 |
| | +diaphragm plasty | 1 | 27 | | 32 | | 31 | |
| Resection left upper lobe | with osteoplastic roof thoracoplasty | 9 | 32 | 23 39 | 32 | 23 44 | 32 | 25 44 |
| | with resection costae 1 5 | 4 | 32 | 24 43 | 38 | 22 53 | 33 | 27 40 |
| | with resection costae 1 7 | 4 | 24 | 17 41 | 18 | 14 22 | 21 | 18 23 |
| | with resection costae 1 8 | 1 | 6 3 | | 9 5 | | 24 2 | |
| | without thoracoplasty | 1 | 16 | | 11 | | 22 | |
| Resection left lower lobe | with osteoplastic roof thoracoplasty | 2 | 22 | 21 23 | 21 | 18 23 | 24 | 23 24 |

FUNCTIONAL RESULT



Right lung Left lung

Fig 8—The functional result found by bronchospirometry at the follow up The percentage given indicates the percentage of oxygen uptake of the remaining lobe after lobectomy in patients with a normal contralateral lung

a cavity in the contralateral upper lobe at the time of operation. Two of these 5 patients have already had segmental resections for removal of the remaining cavitary disease and may come over in the group of "cured" patients at a later date. Two patients are going to be operated upon but the third has been postponed as he has no functional reserve. This is a 36-year-old man with only 28 L. per minute maximal breathing capacity. He has a residual cavity in the left upper lobe under a 7-rib thoracoplasty. After a right upper lobectomy and a 5-rib thoracoplasty, he had still 23 per cent of the total oxygen uptake and 32 per cent of the ventilation on the left lung. The decision to postpone an eventual left upper lobectomy was made before the introduction of the prolonged postoperative artificial ventilation. He had a marked dyspnea walking on level ground but did full time work as a construction engineer.

Two patients with significant apical infiltration have now been working full time for three and four years, respectively, and are negative on direct smear and on guinea pig test of the gastric washing, but the guinea pig test on sputum is positive.

*Patients positive to guinea pig test without x-ray evidence of active disease:* Eight patients had a positive guinea pig test but were negative on examination of the direct smear and the chest; x-ray was without evidence of active disease. Only 3 of these patients had had bilateral disease. Five patients in this group were working full time, one could not work due to retinitis pigmentosa, one did one-half day's work as a clerk (a man with 38 L. per minute maximal breathing capacity). The last patient, a 52-year-old man, had not yet started his heavy work as a blacksmith, one year after surgery.

Two patients had had postoperative tuberculous empyema with tuberculous osteitis and fistulation. One had a revision thoracoplasty, the other seems to heal with local application of antimicrobial therapy. Both patients were negative on direct smear but had positive guinea pig tests. After removal of a suture granuloma in the bronchus, one of the 2 patients is now negative to guinea pig test of sputum as well as to gastric lavage.

## THE FUNCTIONAL RESULT

In order to obtain some information about the functional result after lobectomies with and without different types of thoracoplastics, the oxygen uptake and ventilation on the operated side have been recorded by bronchospirometry one year after operation in a group of patients without present or earlier contralateral pleural or pulmonary disease. The results are given in Table III. It is due to the work by Carlens, first, by the construction of the flexible double lumen catheter and, then, by his actual performance of the bronchospirometric investigation that it has been possible to obtain the permission of a large number of patients to undergo this investigation a year after the operation. From Table III, it can be seen that after removal of the right upper lobe, around 38 per cent of the oxygen uptake remains, regardless of the type and extent of the thoracoplasty. The additional resection of the middle lobe resulted in a loss of a further 12 per cent of the total oxygen uptake. Roughly, the same function, i.e., 26 per cent, remained after removal of upper

In the whole group of 68 patients with earlier contralateral disease as many as 50, or 74 per cent, were negative to guinea pig test one year after the resection. Of the remaining 18, 4 now have already had a bilateral resection. It must therefore be concluded that the contralateral lung has withstood the resection very well, see Table IV.

TABLE IV. THE CONDITION OF THE CONTRALATERAL LUNG AT THE TIME OF OPERATION CORRELATED TO THE RESULT AT THE FOLLOW-UP

| CONDITION OF THE CONTRALATERAL LUNG | "CURE" | RECURRENCE | TOTAL |
|---|---|---|---|
| Normal | 86 | 8 | 94 |
| Old pleuritis | 1 | | 1 |
| Small infiltration | 28 | 9 | 37 |
| Infiltration treated by pneumothorax | 6 | | 6 |
| Cavity closed by conservative treatment | 3 | 1 | 4 |
| Cavity closed by pneumothorax | 12 | | 12 |
| Open cavity | 1 | 8 | 9 |
| Totals | 137 | 26 | 163 |

## SUMMARY

An analysis is made of 301 cases of lobectomies for tuberculosis. Although patients with ventilatory insufficiency were accepted for surgery, the operative mortality was only 2.3 per cent. Of the 163 patients followed up from one to 5 years, it has been gratifying to find 98 per cent with a negative direct smear and 85 per cent with a negative culture and guinea pig test to sputum and gastric washing. Seventy-nine per cent were working full time. The incidence of impaired arm movements, pain, cough, and dyspnea are recorded. The osteoplastic thoracoplasty gave a much better cosmetic result and better arm movements as compared to a thoracoplasty with rib resection. There was, however, a higher incidence of postoperative pain, i.e., 20 per cent instead of 13 per cent in the group who had rib resection. The late functional result has been very good after resection with a space-diminishing procedure. There was usually no significant decrease of the oxygen uptake or ventilation on the operated side after a resection although there was a significant decrease of the maximal breathing capacity. The contralateral side has withstood the resection very well.

## REFERENCES

1. Björk, V. O.: The Surgical Treatment of Pulmonary Tuberculosis, Acta. chir. scandinav. 107: 371, 1954.
2. Björk, V. O.: Thoracoplasty. A New Osteoplastic Technique, J. THORACIC SURG. 28: 194, 1954.
3. Björk, V. O., and Serosoppi, F.: The Arterial Oxygen Tension After Resection With Simultaneous Osteoplastic Thoracoplasty, J. THORACIC SURG. 29: 105, 1955.
4. Björk, V. O.: Opportunité et modalités techniques de la réduction du volume thoracique après lobectomie pour tuberculose, Le Poumon 8: 733, 1955.
5. Björk, V. O., and Engström, C. G.: The Treatment of Ventilatory Insufficiency After Pulmonary Resection With Tracheostomy and Prolonged Artificial Ventilation, J. THORACIC SURG. 30: 356, 1955.
6. Björk, V. O.: The Surgical Treatment of Lower Lobe Tuberculosis, J. THORACIC SURG. 31: 655, 1956.
7. Conklin, W. S., Tuhy, J. E., and Grismer, J. T.: Thoracoplasty Combined With Resection for Pulmonary Tuberculosis, J. THORACIC SURG. 22: 271, 1951.
8. Johansson, L., and Uggla, L. G.: Lobectomy and Pneumonectomy in Tuberculosis, J. THORACIC SURG. 23: 253, 1952.
9. Overholt, R. H., and Wilson, N. J.: Pulmonary Resection in the Treatment of Pulmonary Tuberculosis, Am. Rev. Tuberc. 51: 18, 1945.

and middle lobe as after the lower and middle lobe. On the left side, 32 per cent of the oxygen uptake remained after an upper lobectomy with an osteoplastic thoracoplasty or the resection of the upper five ribs. A significant decrease of function was found when more ribs were resected. Removal of the left lower lobe with an osteoplastic roof thoracoplasty left 22 per cent of the total oxygen uptake on the left side.

It must be noted in one case with phrenic crush and then a total collapse of the right lung for nine years, under a tuberculous empyema in a pneumothorax space, that after decortication and resection of the upper lobe and 5 ribs, 33 per cent of the oxygen uptake and 46 per cent of the ventilation remained on the right side in spite of the fact that it had been collapsed for such a long time.

A comparison of the oxygen uptake and ventilation of each side before and after a resection is only considered to be of secondary interest as (1) the resected lobe is more or less destroyed and without function, and (2) very often a decortication is added which will make the function better after the resection than it was before.

In 34 cases, the function on the operated side was compared before and after lobectomy and the mean difference was for:

| | | |
|---|---|---|
| the oxygen uptake | -2.8% | range + 22% to -23% |
| the ventilation | -0.1% | range + 23% to -36% |
| the vital capacity | -2.6% | range + 16% to -23% |

In the case with the greatest loss of function, i.e., 23 per cent of the oxygen uptake, the right upper lobe was resected in one block together with an infected plomb and the chest wall corresponding to the upper 7 ribs. The greatest improvement of function, i.e., 22 per cent of the oxygen uptake was recorded after a left upper lobectomy with decortication. A gain in oxygen uptake of 11 per cent of the total on the left side was obtained after the removal of a left upper lobe and a thrombus in the pulmonary artery obstructing about 90 per cent of its lumen.

When the maximal breathing capacity was compared before and after resection a significant decrease was found, i.e., 20 L. per minute in men (range +19 to -56) and 13 L. per minute in women (range +22 to -47).

## CONTRALATERAL DISEASE

There were 8 patients with a positive guinea pig test in the 94 who had a normal contralateral lung at the time of operation. Of these, 3 had disease on the operated side and in 4 patients, working full time, the source of the positive guinea pig test could not be detected. Only in one of these 94 patients was an infiltration found in the contralateral lung, which was normal at the time of operation.

In a group of 37 patients having small infiltrations in the contralateral lung at the time of operation, 9, or 25 per cent, had a positive guinea pig test one year after operation.

Nine patients had an open cavity on the contralateral side at the time of operation. In 4 patients the cavity remained open, in 4 bilateral resection had been performed, and in one the cavity was closed.

## CASE REPORT

W. C., a 43-year-old engineer, presented himself on Nov. 18, 1954, with chest roentgenograms demonstrating an abnormal opacity suggestive of tumor. His only symptoms had consisted of a nonpainful tearing sensation with subsequent dull ache in the left shoulder region. He denied dyspnea, cough, hemoptysis, fever, weight loss, weakness, malaise, and night sweats. He had been working actively and playing golf without fatigue up to the time of admission.

*Physical Examination.*—The examination revealed a robust, healthy appearing, well-nourished man in no distress. The temperature was 98.4° F.; pulse was 76; respiration, 18; and blood pressure, 108/74 mm. Hg. Detailed examination revealed no abnormalities except for a small, easily reducible, right inguinal hernia. There was no ptosis of eyelids or muscular weakness. The chest was normal in contour, with excellent respiratory excursion, no areas of dullness to percussion, and normal breath sounds throughout.

Bronchoscopy was entirely normal.

*X-rays.*—Plain film of the chest, dated Feb. 2, 1952, showed normal cardiac and mediastinal silhouette with clear lung fields. Chest films, dated March 6, 1954 (including lateral and oblique views and tomograms), demonstrated a well-circumscribed, 2 by 5 cm., density in the region of the left upper lobe bronchus.

*Operation.*—On Nov. 24, 1954, a left posterolateral thoracotomy was performed under ether-oxygen anesthesia. A plum-sized, soft, encapsulated tumor was found in the anterior mediastinum completely extrapleural and lying on the superior vena cava. There was no evidence of invasion or regional node extension. Satisfactory total excision was accomplished and a frozen section diagnosis of "thymoma" was obtained. Because of the potentially malignant character of this tumor, it was elected to carry out a block dissection of the upper mediastinum cleaning off all tissue between and around the vena cava, aortic arch, innominate, carotid and subclavian vessels, sparing the vagus and phrenic nerves.

*Pathology Report.*—"A 5 × 5 × 2.5 cm. lobulated discord mass with an irregular surface weighs 45 grams. The cut surface is bulging with lobules up to 2 cm. in diameter. Separately submitted is a mass of mediastinal fat. Histologic examination reveals the main tumor to be made up of small and large groups of round cells resembling lymphocytes, areas of clusters of whorls. Hassall's corpuscles are not seen. The entire tumor is enclosed in a fibrous capsule with attached fat. The mediastinal dissection is found to have abundant fatty tissue in which is embedded microscopic foci of thymic tissue and infrequent tiny lymph nodes. A diagnosis of thymoma is submitted; possible malignancy is considered because of the cellularity of the tumor, but in general it is considered benign."

*The Postoperative Course.*—This was completely uneventful. The patient walked without difficulty from the first day and was discharged on the eighth day. He was closely watched for speech slurring, ptosis, muscular weakness, respiratory difficulty, and other indications of myasthenia gravis, but no such signs appeared during his hospitalization.

The patient was seen during an office visit on Dec. 22, 1954; he had walked eight blocks just before examination and showed no evidence of muscular weakness. He felt certain that he had regained strength progressively since operation and that in retrospect there had been no earlier symptoms. On Jan. 1, 1955, he could eat only with difficulty. His symptoms responded dramatically to Prostigmin, thus establishing the diagnosis of myasthenia gravis on January twenty. He required 60 mg. of neostigmine daily to control symptoms for three months. By May, 1955, he had begun gradually to decrease his requirements and by early 1956 was taking no medication.

He was last examined on March 3, 1956, fifteen and one-half months postoperatively. He had walked eight blocks carrying a suitcase and was not fatigued. He showed no evidence of muscular weakness and reported feeling completely normal. He is working full time without difficulty and plays golf.

An x-ray of the chest on March 2, 1956, showed normal mediastinal silhouette and no evidence of tumor recurrence.

# MYASTHENIA GRAVIS SECONDARY TO THYMIC NEOPLASM

## REPORT OF A CASE IN WHICH SYMPTOMS DEVELOPED SIX WEEKS AFTER TOTAL THYMECTOMY

BENSON B. ROE, M.D.
SAN FRANCISCO, CALIF.

THYMIC tumor in association with myasthenia gravis was first noted by Weigert,[1] in 1901. Subsequent reports[2-6] at first suggested that myasthenia gravis occurred with nearly all thymic tumors; other observers,[7-9] however, have found that the disease may be absent and never develops in about 25 per cent of thymic neoplasms. Myasthenia seems less likely to occur with malignant thymic tumors,[8] but the author has had one case with this association and others have been reported.[4, 10, 11] Conversely, it is curious that of all cases of myasthenia gravis, thymic tumor can be demonstrated in about 15 per cent,[8] although some thymic abnormality (hyperplasia) is found in nearly one-half of them.

Thymectomy in the treatment of myasthenia gravis has met with variable success,[8, 12, 13] but it is generally agreed that symptoms are definitely improved by this procedure in many instances, perhaps in more than one-half of the cases.

Careful search of the literature has failed to reveal any description of myasthenia gravis developing after complete removal of thymic neoplasm. Two cases were reported of incompletely removed malignant thymoma in which unmistakable myasthenia complicated the postoperative course. In the case of Fisher and Child,[14] respiratory difficulty developed during operation and the patient died four days later from respiratory complications of the disease. Fershtand and Shaw's case[15] was similar with less certain onset during operation, but postoperative weakness and respiratory embarrassment responded to Prostigmin. This patient improved, later had a temporary remission, and died in one year with recurrent myasthenia gravis and widespread tumor growth. In both of these cases, myasthenia developed coincidentally with or immediately following surgical manipulation and persisted in the presence of remaining thymic tumor tissue. Lack of recognized prior neuromuscular abnormality does not militate against a possible thymic factor in myasthenia; indeed, the course of events in these cases is strong evidence to support the supposed relationship.

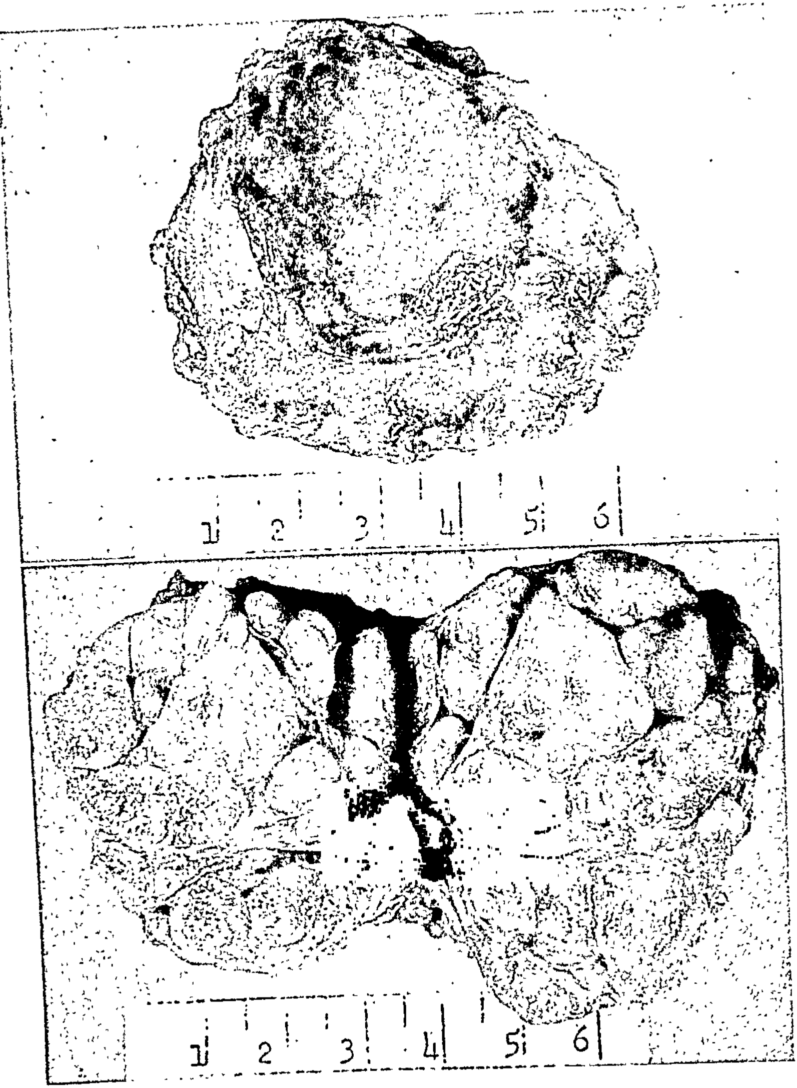
The following case represents what is believed to be a unique instance of myasthenia gravis developing in the absence of thymic tissue and at a time well beyond the patient's convalescence from thymectomy.

---

From the Department of Surgery, University of California School of Medicine, San Francisco, Calif.

Received for publication May 29, 1956.

A.



B.

Fig. 2.—*A*, Gross appearance of tumor. *B*, Gross tumor transected.

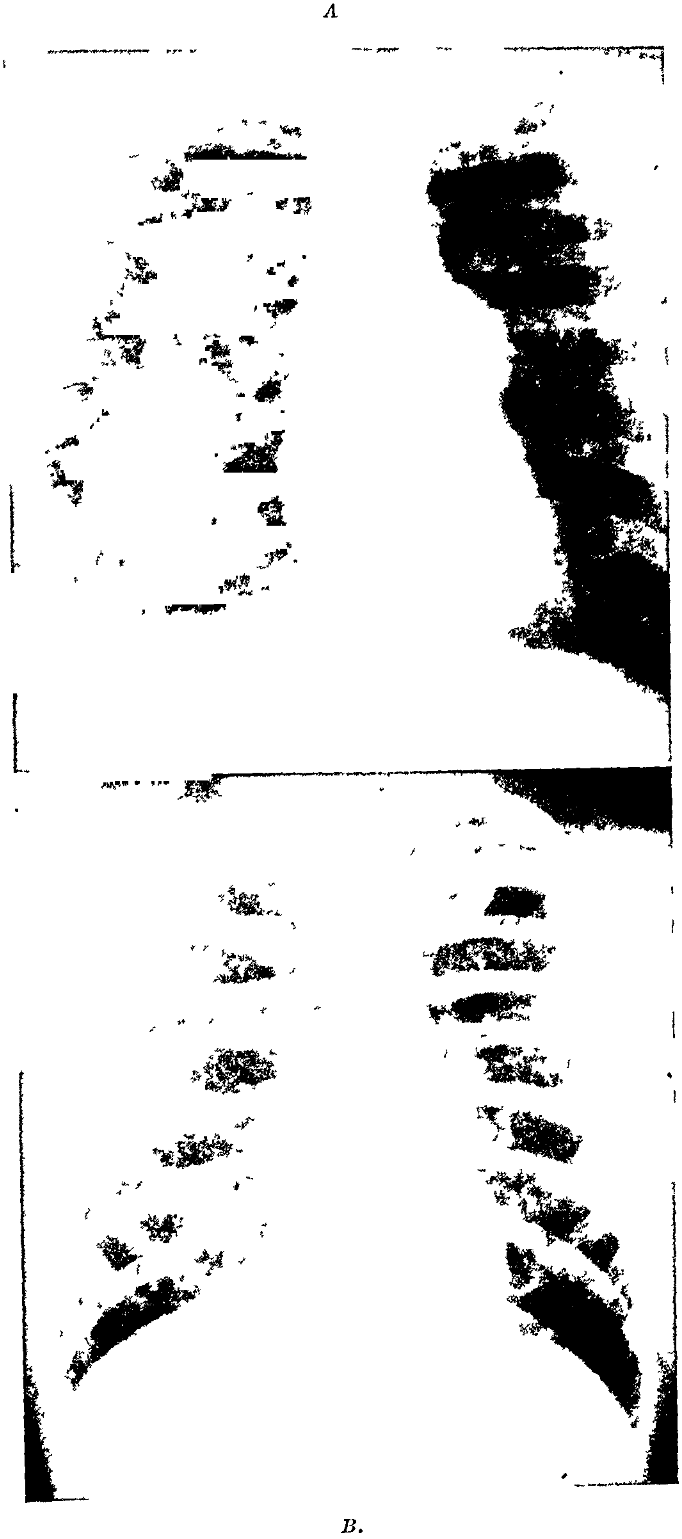


Fig. 1.—*A*, A chest roentgenogram taken preoperatively. *B*, A chest roentgenogram taken three months postoperative.

## DISCUSSION

There seems to be no satisfactory explanation for the unusual course of events in this case Secondary tumor growth or hyperplasia of remaining thymic tissue would have to be postulated by adherents of the thymic origin theory of myasthenia gravis Against this thesis, however, is the complete anterior and upper mediastinal dissection which accompanied the original thymectomy and probably included most of the tissue in which aberrant thymus might occur (though accessory gland tissue has been described both in and below the thyroid gland, laterally in the neck, and behind the sterno mastoid muscle) The clinical course of steady spontaneous improvement with minimal dependence upon Prostigmin is curious, but reflected against experiences with thymectomy for myasthenia gravis this is not unusual Perhaps there is a delayed mechanism between whatever thymic stimulus may occur and the clinical manifestation of the disease If so, this case may represent an instance of fortuitous therapy which failed to interrupt the already initiated sequence of events but did not abort the clinical course of the disease

## SUMMARY

A case of thymic neoplasm is presented in which distinct myasthenia gravis developed six weeks after total thymectomy and radical mediastinal block dissection This is a unique occurrence Related reports in the literature and the problem in general are reviewed

## REFERENCES

1 Weigert, C Pathologisch anatomischer Beitrag zur Erb'schen Krankheit (Myasthenia Gravis), Neurol Centralbl 20 597 601, 1901
2 Bell, E T Tumors of the Thymus in Myasthenia Gravis, J Nerv & Ment Dis 45 130 143, 1917
3 Blalock, A, Mason, M F, Morgan, H J, and Riven, S S Myasthenia Gravis and Tumors of the Thymic Region Report of a Case in Which Tumor Was Removed, Ann Surg 110 544 561, 1939
4 Poer, D H Effect of Removal of Malignant Thymic Tumor in Case of Myasthenia Gravis, Ann Surg 115 586 595, 1942
5 Murray, N A, and McDonald J R Tumors of the Thymus in Myasthenia Gravis, Am J Clin Path 15 87 94, 1945
6 Clagett, O T, and Eaton, L M Surgical Treatment of Myasthenia Gravis, J THORACIC SURG 16 62 80, 1947
7 Heuer, G J, and Andrus, W D The Surgery of Mediastinal Tumors, Am J Surg 50 146 224, 1940
8 Seybold, W D, McDonald, J R, Clagett, O T, and Good, C A Tumors of the Thymus, J THORACIC SURG 20 195 215, 1950
9 Effler, D B, and McCormack, L J Thymic Neoplasms, J THORACIC SURG 31 60 82, 1956
10 Miller, S E, and Redisch, W Malignant Thymoma in Case of Myasthenia Gravis, Ann Int Med 26 440 448, 1947
11 Turnbull, F Removal of Malignant Thymoma in a Case of Myasthenia Gravis Arch Neurol & Psychiat 48 938 945, 1942
12 Blalock, A Thymectomy in the Treatment of Myasthenia Gravis Report of 20 Cases, J THORACIC SURG 13 316 339, 1944
13 Keynes, G Surgery of the Thymus Gland, Brit J Surg 33 201 214, 1946
14 Fisher, J H, and Child, C G, III Myasthenia Gravis Developing Acutely After Partial Removal of a Thymoma, New England J Med 252 891 893, 1955
15 Fershtand, J B, and Shaw, R R Malignant Tumor of Thymus Gland, Myasthenia Gravis Developing After Removal, Ann Int Med 34 1025 1035, 1951

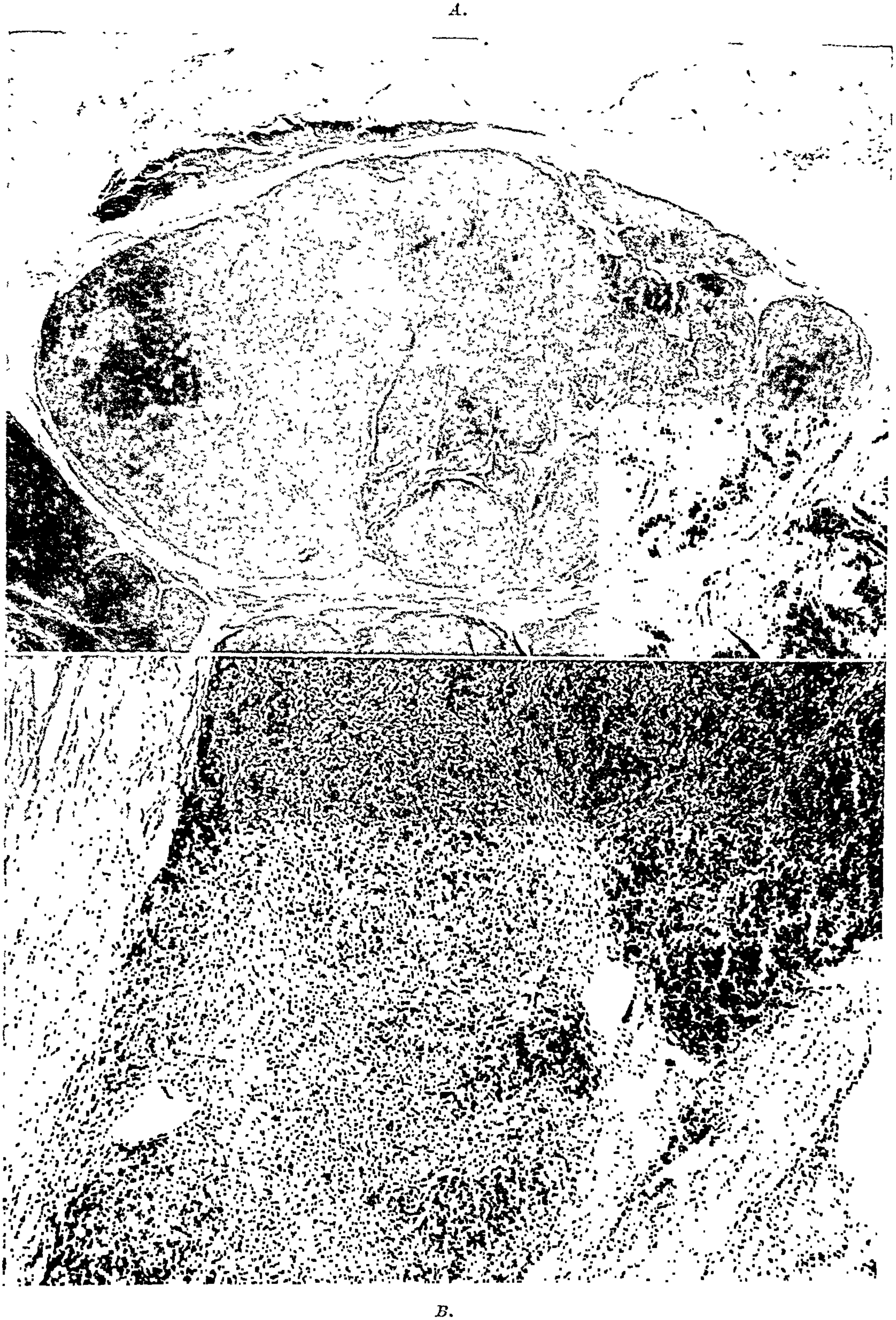


Fig. 3.—*A*, Photomicrograph of tumor. Note encapsulation and trabeculae. (×25; reduced ⅕.)

*B*, Photomicrograph of tumor. (×100; reduced ⅕.)

months. Three of the abscesses had been present for over six months and one of these followed appendectomy two years previously. This chronicity of some cases is well known to surgeons familiar with subphrenic abscess.

The pus obtained in one-third of the cases gave a growth of *Staphylococcus aureus;* in one-third, hemolytic streptococcus, in rather less than a third, *Bacillus coli;* and, in the remainder, a combination of two of these organisms.

*Symptomatology.*—In the common right-sided subphrenic abscess the patient will complain of right posterior lumbar pain and less commonly of pain in the right hypochondrium. In the infrequent occurrence of a left-sided subphrenic abscess, the pain will, of course, be left sided—left upper abdominal if more anterior, left posterior lumbar if more posterior. Only 4 cases in this series were left sided. Sometimes shoulder tip pain is present. Breathlessness may be a feature and results from elevation of the diaphragm restricting the aeration of the lower lobe of the lung. Cough was present in some of the cases. Sputum may be profuse if failure to drain the abscess has allowed it to rupture through the diaphragm into the lower lobe of the lung. There may be a recent history of upper abdominal pain or recent operation on the stomach, duodenum, gall bladder, or appendix.

*Clinical Signs.*—These are characterized by a persistent pyrexia accompanied by an increased respiratory rate; or an elevation of temperature and respiratory rates, three or four days after an operation for, say, the closure of a perforated duodenal ulcer. Tenderness will be present over the twelfth rib and/or below the costal margin. The right lower chest will show diminished movement and there will be impairment to percussion or frank dullness over the right lower lobe while the area of liver dullness may be increased. A discharging upper abdominal wound may be present as a silent witness of a recent operation in which all has not gone well.

X-ray investigation is most helpful. The cardinal findings are elevation of the diaphragm and diminished diaphragmatic movement. Harley (1949)[1] stresses the point that in left-sided subphrenic abscess the mobility of the spleen, stomach, etc., is such as to allow the abscess to enlarge downward; in consequence, elevation of the left diaphragm is much less than in right-sided abscesses. He also makes the useful point that, in right-sided subphrenic abscess, a rise on the right side is an accentuation of the normal elevation and is, in consequence, more obvious. A gas bubble is commonly present under the diaphragm, particularly following the perforation of an abdominal viscus. X-ray films taken of the patient lying on his side will confirm the presence of a fluid level in the abscess. Most patients have a trace of fluid in the costophrenic angle and some may have a quite moderate effusion. As a rule the lower lobe is partly or completely atelectatic and on occasions it is consolidated.

## CASE REPORTS

CASE 1.—The patient had a partial gastrectomy in another hospital three weeks prior to admission to the thoracic unit. Closure of the duodenum had been difficult and the stump leaked on the second postoperative day. At this time, he had developed pain under the right costal margin and he had been tender and guarded in this area. On the third day, he had had pain and tenderness over the right lower ribs posteriorly and by this time he

# SUBPHRENIC ABSCESS

KENNETH FRASER, CH.M. (HONS), F.R.C.S.E., F.R.F.P.S.G.
GLASGOW, SCOTLAND

SUBPHRENIC abscess is not as uncommon as some people believe. The development of this complication should always be borne in mind in all cases in which the diagnosis of an upper abdominal illness is uncertain or in any patient having a stormy convalescence following an upper abdominal operation, such as the closure of a perforated peptic ulcer. Earlier diagnosis and treatment would do much to reduce the not inconsiderable mortality of this condition and it is for this reason that it seemed justifiable to put on record 30 cases seen personally by the author and the results obtained in 25 of these, following operation.

An analysis of the primary lesions causing subphrenic abscess is shown in Table I.

TABLE I. PRIMARY LESION LEADING TO SUBPHRENIC ABSCESS

| | |
|---|---|
| Duodenal ulcer perforation (sutured) | 9 |
| Leak or suspected leak from duodenal ulcer | 6 |
| Acute appendicitis (appendectomy) | 3 |
| Acute cholecystitis or gallstones | 3 |
| Wounds in region of liver | 3 |
| Boils or carbuncles | 2 |
| Postcholecystectomy | 1 |
| Postgastrectomy | 1 |
| Liver abscess | 1 |
| Puerperal sepsis | 1 |
| Total | 30 |

It will be noted that nearly one-third of the cases followed the closure of a perforated duodenal ulcer, and one-half, if one adds to these, the cases in which a duodenal ulcer had leaked or was suspected to have leaked. Three abscesses followed acute appendicitis, in spite of appendectomy, and three were associated with acute cholecystitis or cholelithiasis. A further 3 cases resulted from war injuries to the liver; one of these patients was a naval rating, serving on an aircraft carrier, who was hit in the abdomen by the wing of a landing airplane and sustained rupture of the liver. Two cases gave a history of recent boils or carbuncles. Failure to inquire about this type of recent infection may account for some of those reported cases in which no cause for the subphrenic abscess was found. One case each occurred following cholecystectomy, gastrectomy, liver abscess, and puerperal sepsis.

The abscess may develop within a few hours of the onset of the primary lesion or it may be of quite insidious onset and only diagnosed after many

---

This article is based on a paper delivered to the Society of Thoracic Surgeons of Great Britain and Northern Ireland in November, 1955.

Received for publication May 15, 1956.

Case 2.—The patient, a 76-year-old man in poor general condition, was admitted with right upper abdominal pain which was thought to be due to acute cholecystitis. At operation pus was obtained from the subphrenic space in the region of Morison's pouch (Figs. 4 and 5).

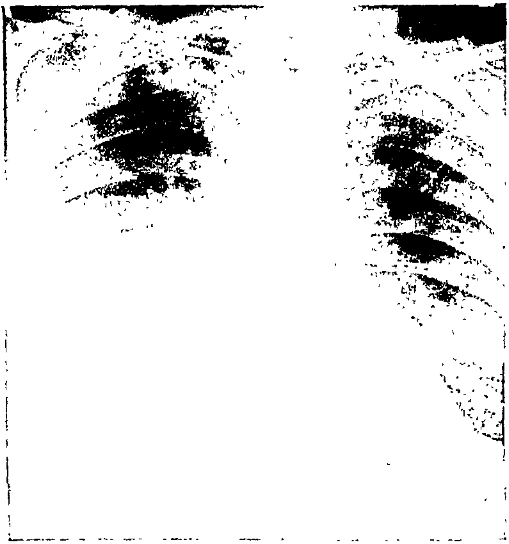


Fig. 4.

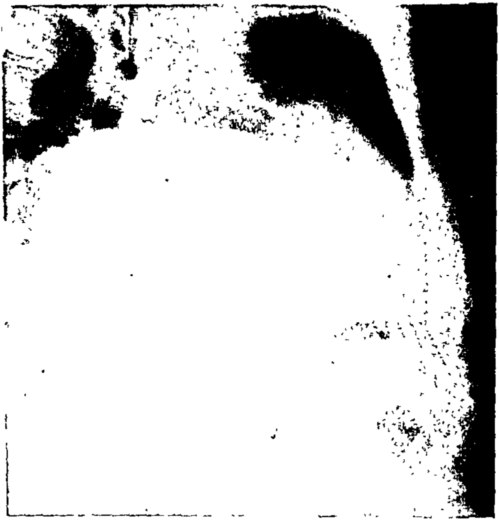


Fig. 5.

Figs. 4 and 5.—Case 2. Marked elevation of the diaphragm in subphrenic abscess resulting from acute cholecystitis.

was running an intermittent pyrexia which responded, only for a time, to penicillin. There were dullness and diminished movement over the right lower lobe. The x-ray films showed elevation of the right diaphragm over a gas bubble; the lower and middle lobes were partly atelectatic (Figs. 1 and 2). This patient was drained anteriorly and Fig. 3, taken some days later, shows the drainage tube in situ with Lipiodol instilled.

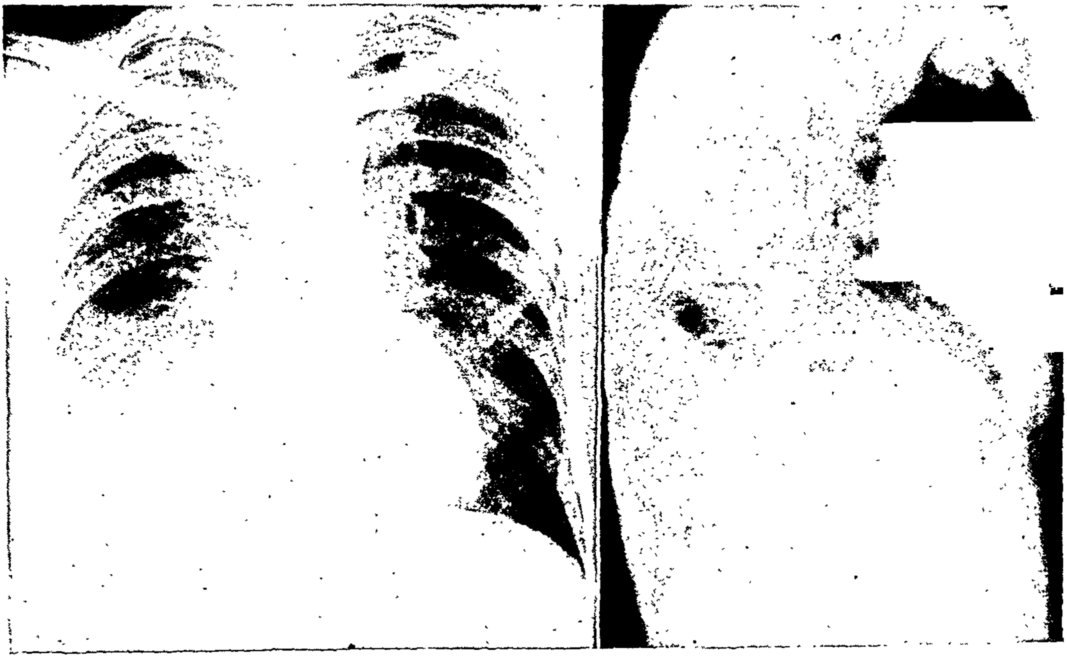


Fig. 1. Fig. 2.

Figs. 1 and 2.—Case 1. Elevation of the right diaphragm over a gas bubble. There is partial atelectasis of the lower and middle lobes.

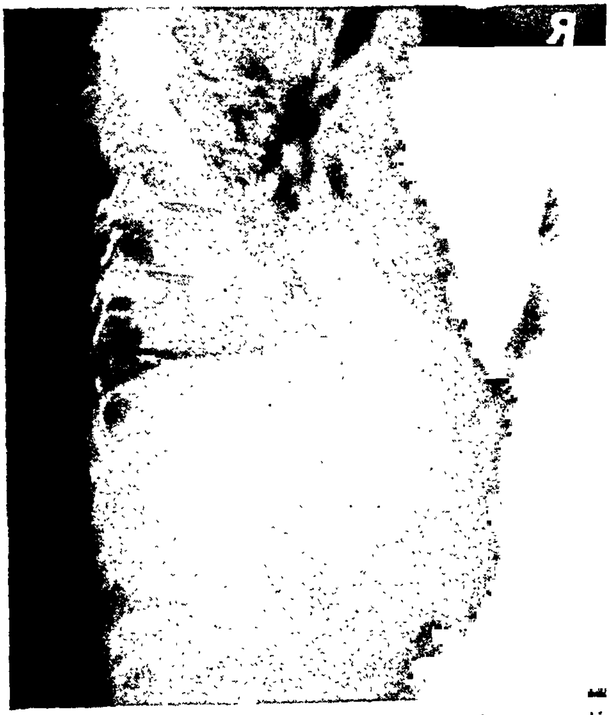


Fig. 3.—Case 1. Lipiodol filled drainage tube several days after operative drainage by the anterior route.

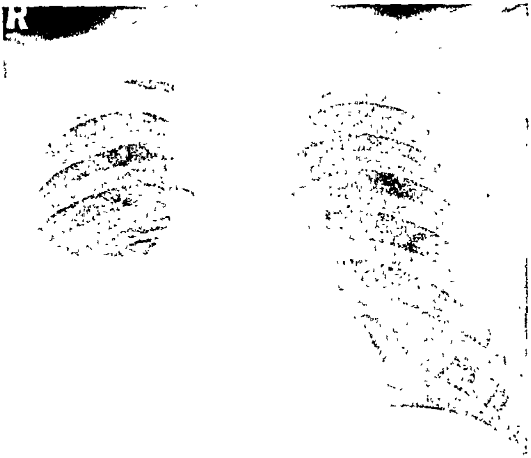


Fig. 8.

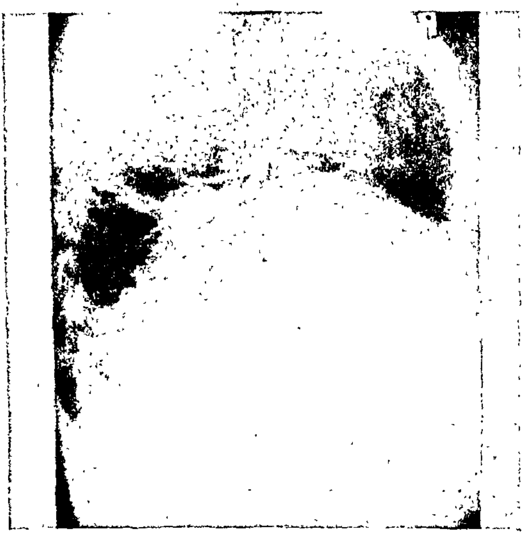


Fig. 9.

Figs. 8 and 9.—Case 3. Bronchogram confirms the elevation of the diaphragm and shows the upward displacement of the atelectatic lower and middle lobes.

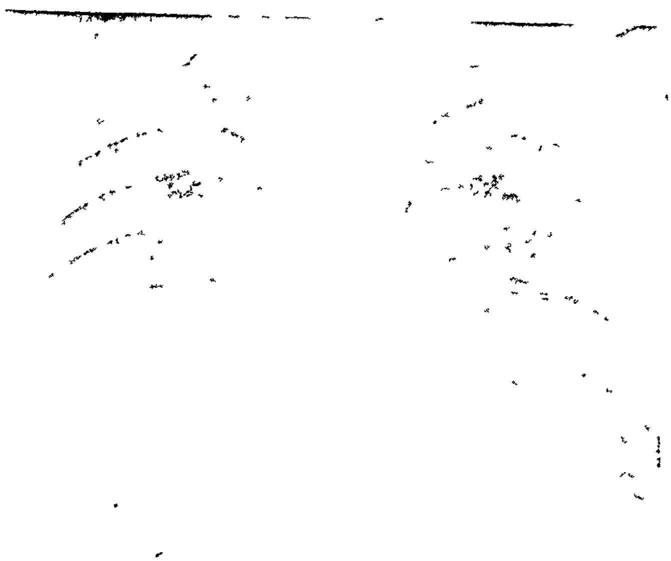


Fig. 6

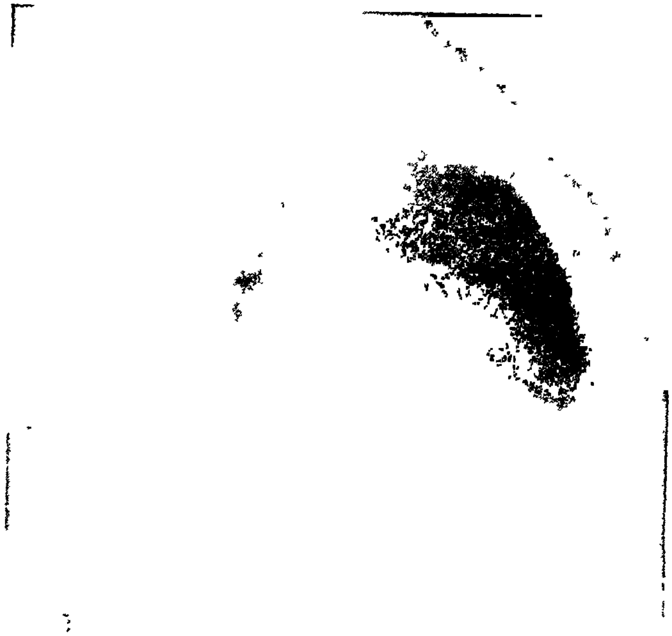


Fig 7.

Figs. 6 and 7.—Case 3. Marked elevation of diaphragm due to a subphrenic abscess developing subsequent to the operation of cholecystectomy.

the sitting up position, it also has the merit of a mortality rate of nearly half that of drainage by the transserous route (Harley, 1949) On rare occasions when the abscess is obviously presenting anteriorly, drainage from the front may be preferred

### RESULTS

Table II shows the results in the 30 cases of this series It will be seen that subphrenic abscess is a condition which carries quite a fair mortality In 25 cases drainage was used, 3 of these patients died, giving a mortality of 12 per cent In 5 patients, drainage was never established and, of these, 2 died, giving a mortality of 40 per cent In the total of 30 cases there were five deaths—an over all mortality of 16 6 per cent

TABLE II THE OUTCOME IN 30 CASES OF SUBPHRENIC ABSCESS

| | NUMBER OF CASES | NUMBER OF DEATHS | MORTALITY (%) |
|---|---|---|---|
| Evacuation of abscess and drainage | 25 | 3 | 12 |
| Not drained | 5 | 2 | 40 |
| Total of 30 cases with 5 deaths | | | 16 6 |

In the series of 182 cases collected by Harley (1949), the mortality for the cases in which drainage was used was 26 6 per cent, and for those without drainage, 83 per cent, giving an over all mortality of 40 1 per cent While the figures in this series compare very favorably with the collected series of Harley (1949), the mortality is still far too high

Of the five deaths, four resulted largely because the diagnosis was delayed too long and the provision of drainage was either inadequate or too late Admittedly, 2 of these patients were old men in their middle seventies, but one was a healthy man, aged 25, in whom the diagnosis was so protracted that metastatic brain abscesses developed and drainage was never established

The fifth case was masked in that the perforation of the duodenal ulcer occurred into the lesser sac

### CONCLUSION

It is generally true to say that it is only necessary for the thoracic surgeon to hear the clinical history of a case to be able to suggest the diagnosis Subphrenic abscess should be suspected in any patient recently submitted to an upper abdominal operation, particularly a perforated peptic ulcer, if he has continuing upper abdominal and low back pain accompanied by swinging temperature It is my experience, however, that surgeons, particularly general surgeons, are not sufficiently aware of the possibility and frequency of the occurrence of this complication Also, once having made a firm diagnosis of subphrenic abscess, they are very liable to hold a "watching brief" over what may well turn into "watching disaster" Delay in treatment or failure to achieve adequate drainage by anterior or lateral approach may well result in the abscess bursting through the diaphragm, with consequent bronchiectasis Delay, as we have seen, may result in the patient's death

CASE 3.—This patient, a 42-year-old man, had had a cholecystectomy in another hospital ten months prior to admission to the thoracic unit. He had had a stormy convalescence and the wound burst. The right lung collapsed and he had developed a pleural effusion. Six months later, he developed recurring fever, and pain in the right upper quadrant of the abdomen, accompanied by shoulder tip pain. One month prior to transfer to the thoracic unit, an attempt was made to drain the abscess but only a little pus was obtained. Posteroanterior and lateral x-ray films showed a very high diaphragm with collapse of the middle and lower lobes of the lung (Figs. 6 and 7). In this type of chronic case, the diagnosis may be difficult and, if doubt exists, bronchography will make the situation abundantly clear as to whether the shadow is, in fact, due to a raised diaphragm or not (Figs. 8 and 9). Three and one-half pints of pus were emptied from this abscess and Fig. 10 shows Lipiodol instilled into the sinus some days after drainage by the standard posterior approach.

Fig. 10.—Case 3. An x-ray which shows Lipiodol instilled into the posteriorly placed drainage wound.

*Treatment.*—One agrees with Hunt (1948)[2] that diagnostic aspirations should be avoided for fear of spreading infection to pleura or peritoneum. Operation to establish drainage should be performed if the subphrenic abscess is of any size and if, in spite of antibiotic treatment, the pyrexia continues and is accompanied by pain and tenderness. The usual posterior incision is made over the twelfth rib and a sufficient length of the rib is excised to allow the operator's hand to enter the wound. If the pleural reflection presents, it is displaced upward and the diaphragm is traversed, the upper surface of the liver being easily reached. A very wide stiff drainage tube is used and should be left unshortened for several days. The posterior approach has the advantage of being extraserous and of giving dependent drainage to a patient in

# INTRATHORACIC LIPOMAS

WILLIAM T. WILLIAMS, M.D.,* AND WILLARD H. PARSONS, M.D.**
JACKSON, MISS.

JUDGING from the number of cases reported in the literature, the care of 2 patients with an intrathoracic lipoma during a five-year period by a single individual would be an unusual circumstance. Keeley and his associates[1] gathered 57 cases from the literature and added a case of their own. Since that time, 8 cases have been added,[2-9] bringing the total to 66. In this communication, 2 cases of intrathoracic lipoma are presented† along with a brief discussion of the tumor.

## CASE REPORTS

CASE 1.—L. H., a 32-year-old Negro man, was first seen in January of 1950 because an abnormality of the right lung field had been detected three months previously on a routine chest x-ray. He was without symptoms and gave a history of smoking one package of cigarettes daily.

No abnormalities were demonstrated on physical examination and, except for a moderate anemia, all laboratory reports were normal. Roentgenograms of the chest (Fig. 1) showed a semicircular mass overlying the upper right lung posteriorly. The roentgenologist's impression, after reviewing the stereoscopic films, was that the mass lay in an extrapleural position, located in the soft tissues along the medial border of the scapula.

Following correction of the anemia, exploratory thoracotomy was carried out six days following admission. Elevation of the scapula revealed no evidence of the tumor mass between it and the thoracic cage as had been indicated by the roentgenologist. When an intercostal incision was made between the fourth and fifth ribs and the pleura opened, a soft cystic tumor about the size of a hen's egg was found in the pleural space adjacent to the fifth rib and the posterior axillary line. Clinically and histologically, the tumor was a lipoma. The tumor was excised and the patient recovered uneventfully (Fig. 2) and is in good health four years following operation.

CASE 2.—V. A., No. 61018, a 55-year-old Negro man, was admitted to the Veterans Hospital Jan. 29, 1955, complaining of chronic cough, wheezing, shortness of breath, and a slightly productive cough of several years' duration. He had been investigated in 1949 because of recurring precordial pains with radiation to the right shoulder and right arm. However, no abnormalities were demonstrated at this time and he was discharged without a positive diagnosis.

Physical examination on the present admission revealed slight dyspnea at rest and a pulse rate of 92 per minute. In addition, dry expiratory wheezes were heard over the lower lung fields, bilaterally. A routine chest film (Fig. 3) revealed a large ovoid density in the left base, projecting approximately 2 cm. above the level of the diaphragm. Fluoroscopic examination showed the mass to lie far posteriorly in the costophrenic sinus. It was suggested at that time by the roentgenologist that the mass might represent a herniation of a subdiaphragmatic organ and, for that reason, additional roentgenograms following pneumoperitoneum were obtained, demonstrating the mass to be attached to the diaphragm and to lie above the diaphragm (Fig. 4). It was thought by the roentgenologist

---

From the Department of Surgery, University of Mississippi Medical Center and the Veterans Hospital, Jackson, Miss., and the Vicksburg Hospital, Vicksburg, Miss.

Received for publication May 31, 1956.

*Instructor in Surgery, University of Mississippi School of Medicine, Jackson, Miss.

**Clinical Associate Professor of Surgery, University of Mississippi School of Medicine. Chief of Surgery, Vicksburg Clinic and Vicksburg Hospital, Vicksburg, Miss.

†Case 1 previously reported. The Surgical Management of Intrathoracic Lesions, The Mississippi Doctor 31: 387, 1954.

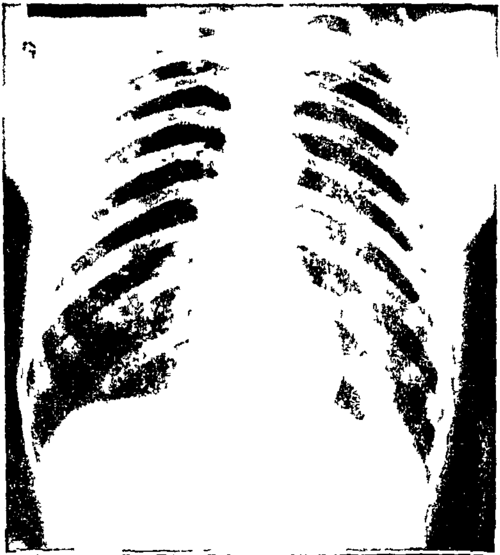
Subphrenic abscess should be thought of if the circumstances just discussed are found, examination and investigation for it ought to be instituted, and radical drainage should be established forthwith if the abscess is of large dimensions or is found to be unresponsive to early conservative treatment.

It is true to state in this complication of subphrenic abscess as in all conditions in which a serious or even fatal complication may occur—there is some excuse to have looked and found nothing, there is no excuse for not having looked at all.

## REFERENCES

1. Harley, H. R. S.: Subphrenic Abscess, Thorax 4: 1, 1949.
2. Hunt, R. S.: Subphrenic Abscess, Brit. J. Surg. 36: 185, 1948.

Fig 3



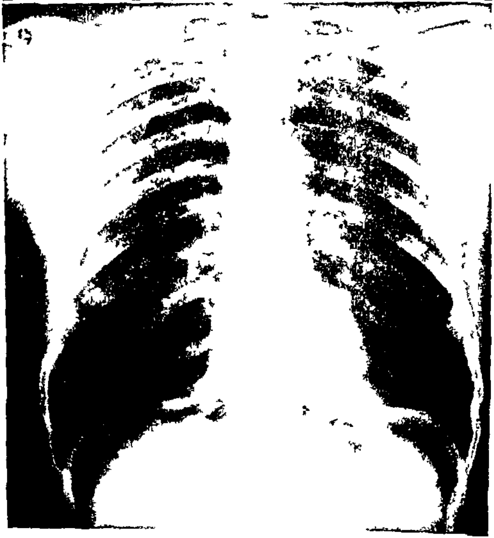


Fig 4

Fig 3 —Rounded density projecting above diaphragm in left cardiophrenic angle
Fig 4 —Following pneumoperitoneum

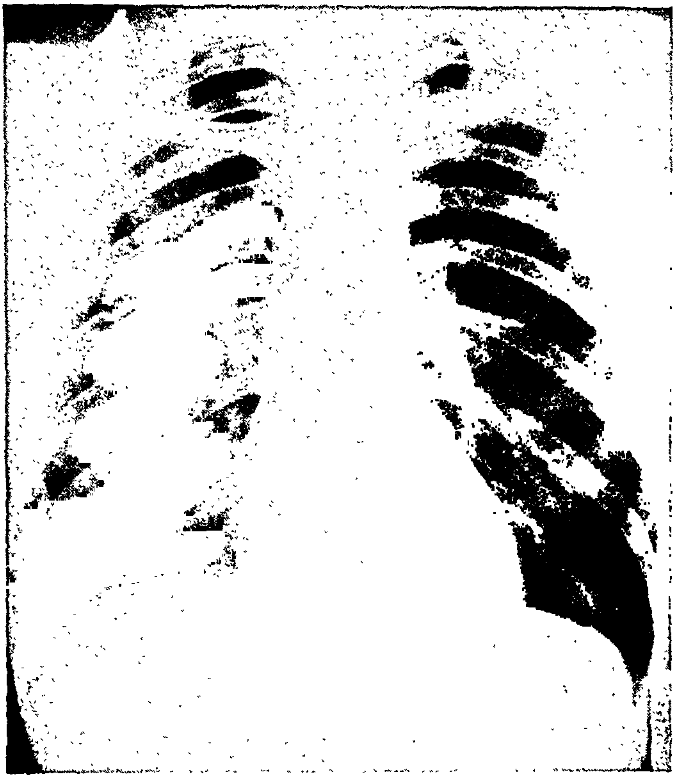


Fig. 1.—Smooth-rounded density overlying right anterior first and second ribs.

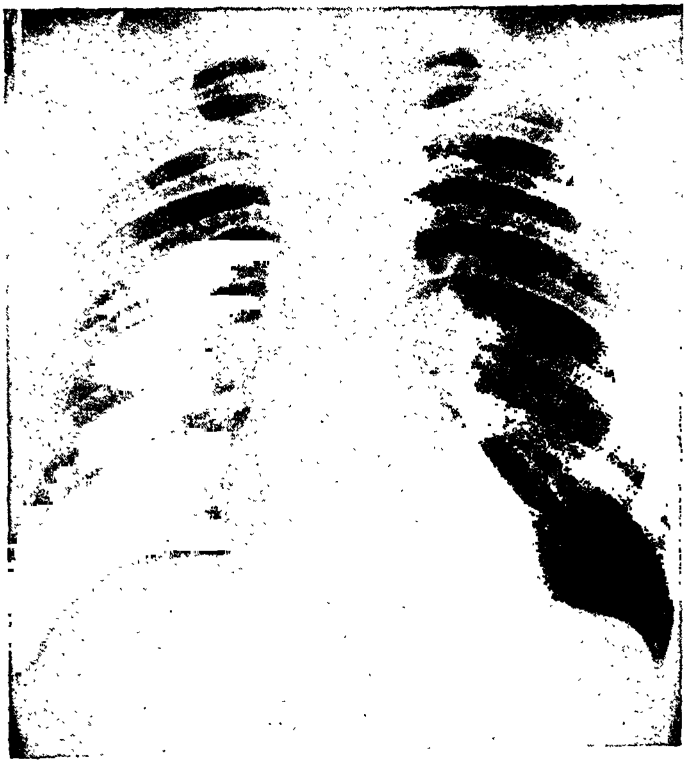


Fig. 2.—Postoperative film.

that the mass represented a benign tumor, most likely a lipoma. Because of the unknown nature of the tumor, the left chest was explored through the bed of the resected eighth rib. A 7 cm. tumor, having all the characteristic appearances of a lipoma, was easily demonstrated in the costophrenic sinus at the most posterior part of the left diaphragm (Figs. 5 and 6). After the pleura overlying the tumor had been incised, it was demonstrated that the lipoma arose from the properitoneal fat and extended through the diaphragm, separating the muscle fibers of that structure. The tumor was excised and the diaphragm repaired. The postoperative course was uncomplicated except for accumulation of fluid following removal of the thoracotomy tube. This was corrected by two aspirations and the patient was discharged with essentially the same symptoms as he presented on admission.

## DISCUSSION

The classifications of lipoma previously proposed have been related to the anatomic site of the tumor.[1]

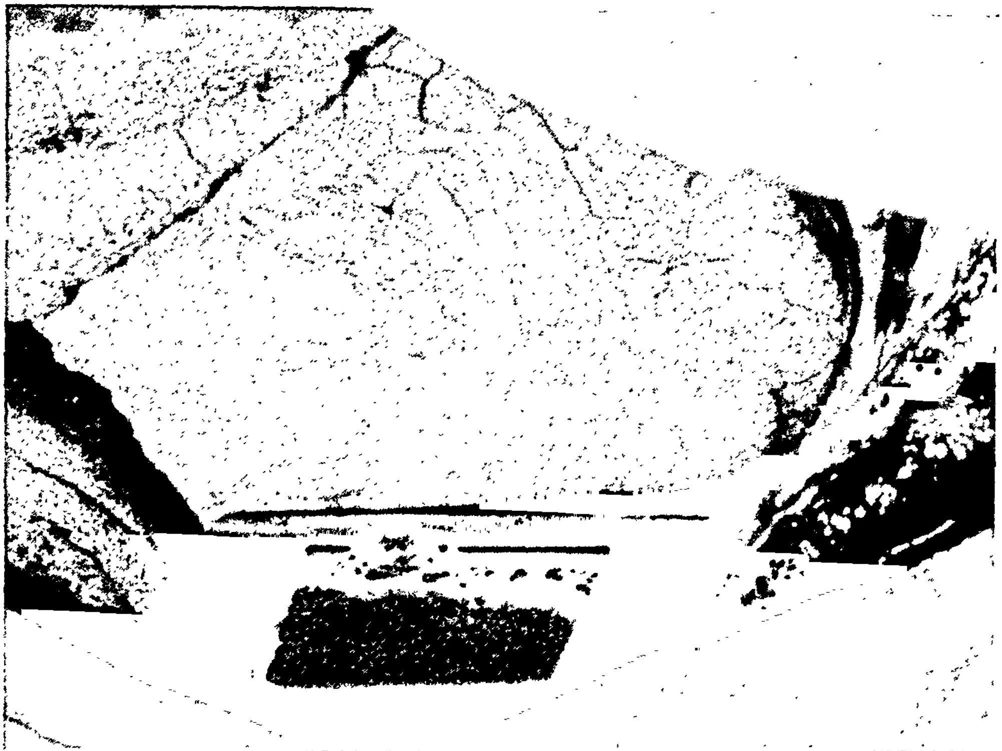
1. Intrathoracic lipomas: those lying entirely within the thoracic cage.
2. Hourglass thoracic lipomas: those having both intra- and extrathoracic portions.
    a. Cervicomediastinal, passing from the mediastinum into the neck.
    b. Transmural, passing through the chest wall—usually an intercostal space, rarely through the sternum.

In addition to these locations, lipomas may arise intrabronchially[10, 11] and from the external surface of the heart.[12] In addition to this anatomic classification, a histologic one might be considered, although this would necessarily include cases other than pure lipomas such as we are presenting here. This classification would include liposarcomas,[13] hibernomas,[14] and the somewhat controversial thymolipomas.[15] Rubin and Mishkin[15] have suggested that thymolipomas are actually lipomas per se which have their origin in thymus gland fat and carry remnants of the thymus with it as the bulk and weight of the tumor cause it to gravitate to the more inferior confines of the mediastinum.

The symptoms and physical findings presented by the patient depend upon the size and location of the tumor and, as would be expected, asymptomatic lipomas are frequently discovered on routine chest films. When this is the case in the differential diagnosis, one should keep in mind that lipomas have been confused with cardiac enlargement[8] and, in addition, they must be differentiated from those tumors which frequently arise in the location occupied by the lipoma in question. Keeley[1] pointed out that the good general condition of the patient with a lipoma of such size and configuration as to represent a far advanced malignant process on chest x-ray was an important point in the differential diagnosis of the tumor. In addition, Heuer and Andrus[16] have described the x-ray of intrathoracic lipomas as being characteristic, suggesting that the difference in specific gravity between fat and fluid produces a relatively clear peripheral zone in the lipoma which is not found in fluid. Other diagnostic measures which have been employed include pneumothorax, pleuroscopy, and needle aspiration. The use of pneumoperitoneum in our second case was useful in differentiating the tumor of the diaphragm from a diaphragmatic hernia.

Despite the many diagnostic aids at our disposal it is frequently impossible to determine which tumors are lipomas and which are not. Along this

Fig. 5.



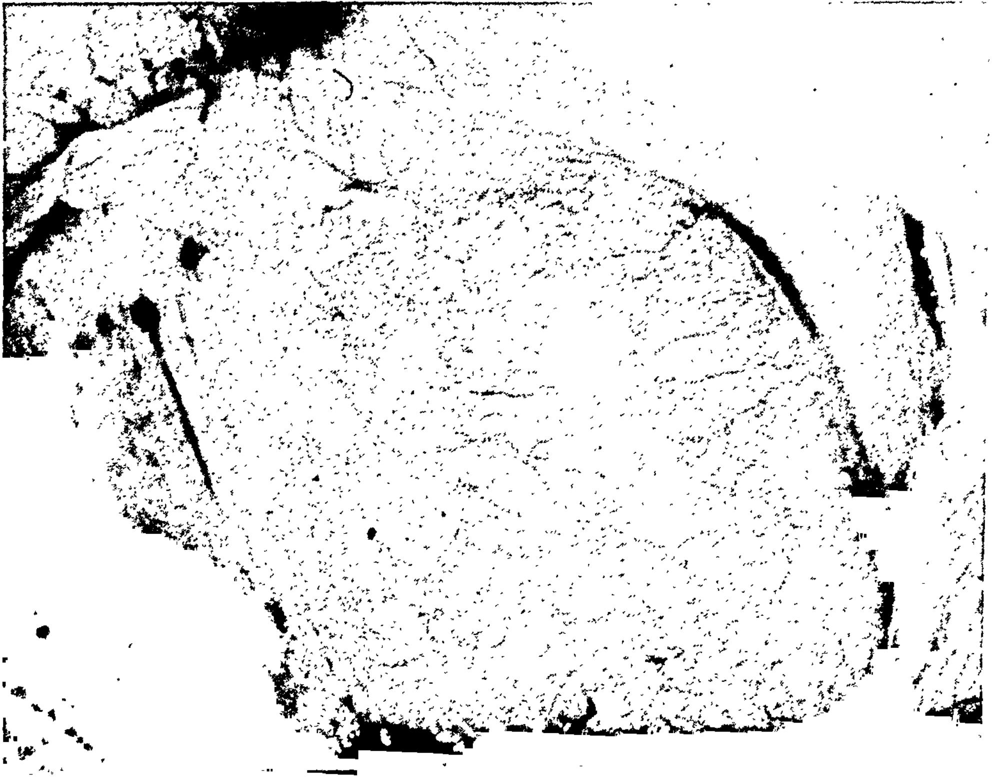


Fig. 6.

Fig. 5.—Smooth-rounded tumor in posterior medial part of diaphragm.
Fig. 6.—Same as Fig. 5, but with diaphragm retracted anterior and laterally.

# BRONCHOPULMONARY SEQUESTRATION

## SOME CLINICAL ASPECTS WITH REPORT OF SIX CASES

CHARLES T. PINNEY, MAJOR, MC, USA,* AND
JOHN M. SALYER, COLONEL, MC, USA**
DENVER, COLO.

SINCE 1951, 6 cases of intralobar bronchopulmonary sequestration have been diagnosed and operated upon at Fitzsimons Army Hospital. These congenital lesions occurred in young soldiers varying in age from 17 to 24, and 5 of the 6 cases were asymptomatic. Nevertheless, it has been stated that most patients with that condition give a history of repeated pulmonary infections with chills, fever, productive cough, and roentgenographic abnormalities in the lower lung fields.[1]

This condition has received a great deal of attention in the medical literature since 1946 when Pryce published his first paper on the subject.[2] Anomalous arterial supply to the lungs, however, was first described in *1802* by Maugars, a French medical student.[3] Descriptions of surgical procedures in over 100 cases have appeared in the literature. This entity is important to the surgeon because it represents a hazard to those not familiar with the condition; under such a circumstance, technical difficulties may arise as dissection is performed in the vicinity of the anomalous artery. No technical errors have occurred during the surgical treatment of our small series of patients—due largely to the roentgenographic findings and diagnostic implications. Excellent reviews of this interesting anomaly have been published since 1950 by Bruwer,[1] Wyman,[4] Boyd,[5] and Cole,[6] and their associates. The reader is referred to these publications for detail concerning the embryology, pathogenesis, and pathology.

The purpose of this paper is to report 6 more cases of intralobar sequestration, 5 of which were discovered by routine roentgenograms. The diagnosis was established in one case by retrograde aortography. Three of the 6 cases were seen within a period of five months in 1955. We wish to stress the importance of laminagraphy as an additional diagnostic aid and to re-emphasize that this condition probably exists in the asymptomatic state more often in young adults than in the symptomatic state.

---

From the Departments of Medicine and Surgery, Fitzsimons Army Hospital, Denver 8, Colo.

Received for publication June 11, 1956.

*Present address: The Olean Medical Group, 201 South Union St., Olean, N. Y.

**Chief, Thoracic Surgery Service, Fitzsimons Army Hospital, Denver, Colo.

line, it is interesting to note the reported number of cases of liposarcoma and to compare that figure with the number of lipomas found in the literature. Storey and Knutson[13] gathered from the literature five solitary liposarcomas of the mediastinum and 5 cases of multiple liposarcomas. When these 10 cases of liposarcoma are numerically compared with the 57 cases of lipoma which had been reported at about that time, one wonders whether the incidence of sarcomatous degeneration in a thoracic lipoma may be higher than in other locations. However, it may well be that these figures are distorted by the fact that asymptomatic intrathoracic lipomas are not discovered and, hence, do not find their way into the literature.

In any event, the treatment of choice is exploratory thoracotomy and excision of the tumor. Once the chest is open and the tumor is in view, there is usually little difficulty in identifying the lipoma as such and affecting its removal. When dealing with especially large lipomas, or other bulky intrathoracic tumors for that matter, it must be appreciated that compensatory space adjustments have occurred during the slow growth of the tumor.[17, 18] When the pleural cavity is opened, these compensatory adjustments are disturbed and potentially lethal cardiorespiratory problems arise which must be immediately recognized and rapidly corrected.

## SUMMARY

Two cases of unusually located intrathoracic lipomas are reported and the subject briefly discussed.

## REFERENCES

1. Keeley, J. L., Gumbiner, S. H., Guzauskus, A. C., and Rooney, J. A.: Mediastinal Lipoma: The Successful Removal of 1,700 Gram Mass, J. Thoracic Surg. 25: 316, 1953.
2. Fine, E. H., and Joubert, G. A.: Intrathoracic Lipoma; Report of a Case, South African M. J. 27: 809, 1953.
3. Cesanelli, A.: Lipoma Intratorácico, An. Cir. 17: 166, 1952.
4. Barré, E., Danrigal, A., Maruelle, R., and Rolland, L.: Lipome thoracique antéro-inférieur droit, J. franç. méd. et chir. thorac. 8(5): 501, 1954.
5. Bonniot, A., and Barrié, Brancato: Sur un cas de lipome du médiastin postérieur, Lyon Chir. 48:(4) 487, 1953.
6. Parsons, W. H., Hamernik, F. J., and Williams, William T.: The Surgical Management of Intrathoracic Lesions, Mississippi Doctor 31: 387, 1953-54.
7. Crutcher, R. R., and Plott, C. L.: Mediastinal Lipoma, J. Thoracic Surg. 27: 261, 1954.
8. Gottlieb, A. M., Baer, L. J., and Jordan, P., Jr.: Mediastinal Lipoma Simulating Cardiac Enlargement, J. A. M. A. 152: 908, 1953.
9. Goyer, R., Bernou, A., and Tricoire, J.: Un nouveau cas de lipome du méediastin, J. franç. méd. et chir. thorac. 8(6): 678, 1954.
10. McCall, R. E., and Harrison, W.: Intrabronchial Lipoma, A Case Report, J. Thoracic Surg. 29: 317, 1955.
11. Brewin, E. G.: A Case of Lipoma of the Bronchus Treated by Transpleural Bronchotomy, Brit. J. Surg. 40: 282, 1952-53.
12. Maurer, E. R.: Successful Removal of Tumor of the Heart, J. Thoracic Surg. 23: 479, 1952.
13. Storey, C. F., and Knutson, K. P.: Liposarcoma of the Mediastinum, J. Thoracic Surg. 22: 300, 1951.
14. Peabody, J. W., Ziskind, J., Buechner, H. A., and Anderson, A. E.: Intrathoracic Hibernoma, New England J. Med. 249: 329, 1953.
15. Rubin, M., and Mishkin, S.: The Relationship Between Mediastinal Lipomas and the Thymus, J. Thoracic Surg. 27: 494, 1954.
16. Heuer, G. J., and Andrus, W. DeW.: The Surgery of Mediastinal Tumors, Am. J. Surg. 50: 146, 1940.
17. Clagett, O. T., and Hausmann, P. F.: Huge Intrathoracic Fibroma, Report of a Case, J. Thoracic Surg. 13: 6, 1944.
18. Barett, N. R., and Barnard, W. G.: Some Unusual Thoracic Tumors, Brit. J. Surg. 32: 447, 1945.

inferior pulmonary ligament, a large artery was noted to enter the thoracic cavity through the diaphragm and enter the lower lobe. A right lower lobectomy was performed without complications.

At gross pathologic examination, multiple cysts filled with gelatinous material were found. There were no demonstrable bronchial communications with these cysts. The *anomalous artery measured 6 mm. in diameter and supplied the cystic portions of the lung.* Microscopic examination revealed that the cysts were lined by columnar type epithelium. The pulmonary parenchyma showed remnants of alveolar architecture, considerable fibrosis and other chronic inflammatory changes were noted.

Case 2.—A 24-year-old Negro corporal was transferred to Fitzsimons Army Hospital on Dec. 9, 1952, with an undiagnosed mass in the left lung.

The patient had been entirely asymptomatic and a routine physical examination and chest roentgenogram in September, 1952, revealed a homogeneous rounded mass in the left lower lung field. The past medical history was negative and there was no history of pneumonia. The physical examination was within normal limits.

The laboratory findings revealed that the hemogram, the urinalysis, and the sedimentation rate were normal. Tuberculin test (purified protein derivative No. 1) was positive. The histoplasmin and coccidioidin skin tests were slightly positive. Culture of the sputum and gastric contents for acid-fast bacilli was negative on several occasions.

A chest roentgenogram revealed a 2 cm. rounded mass of homogeneous density in the medial portion of the lower lung field on the left (Fig. 2). A lateral x-ray localized this area to the posterobasal segment. Review of x-rays which were taken prior to this patient's induction into military service revealed that this lesion had been present since November of 1950 and had been unchanged since that time. A bronchogram demonstrated the presence of a large emphysematous area with distortion and displacement of all basilar bronchi (Fig. 3).

A diagnostic thoracotomy was performed on March 19, 1953. The lateral and posterior basal segments of the left lower lobe appeared pink and showed no visible anthracotic pigment. Located in the upper portion of the lateral and posterior basal segments was a cystic mass which measured approximately 3 cm. in its greatest diameter. In the pulmonary ligament, an artery was discovered which came from below the diaphragm and measured 5 mm. in diameter. A left lower lobectomy was performed without complications.

On gross pathologic examination the posterobasal segment was noted to be quite pink and devoid of visible anthracotic pigment. A 3 cm. mass was palpated in the posterior aspect of the posterobasal segment. In the medial portion of the posterobasal segment, a large 3 cm. cystic mass was demonstrated and was filled with a greenish-gray mucoid material. There was no bronchial communication. An anomalous artery was noted to run just lateral to the cystic mass and divide into several branches in that area. The cyst was irregular and possessed a very thin, grayish-white wall.

Microscopic examination revealed that the cyst was lined with columnar type epithelium and the pulmonary parenchyma revealed evidence of fibrosis and inflammation.

Case 3.—A 22-year-old white male sergeant was admitted to Fitzsimons Army Hospital on Nov. 27, 1953, for evaluation of an infiltrate in the right lower lobe.

The present illness began in October, 1953, and was characterized by an upper respiratory infection which was associated with a mild cough productive of small amounts of white sputum with occasional streaking of blood. This infection lasted for approximately two weeks. The patient was treated with penicillin and Aureomycin which caused a disappearance of all his symptoms and he had remained in good health thereafter. A chest roentgenogram made at this time revealed an area of infiltration in the right cardiophrenic angle. Tuberculin (PPD) skin tests were negative. Cultures of the sputa and gastric contents for acid-fast bacilli were negative.

The past history is significant in that the patient had had two previous attacks of pneumonia, one in 1949 and one in 1950.

The physical examination was within normal limits.

## CASE REPORTS

Case 1.—A 21-year-old white enlisted man was transferred to Fitzsimons Army Hospital on April 7, 1951, with a diagnosis of cystic disease of the lung. His symptoms began in March, 1951, and consisted of the sudden onset of right sided chest pain which was aggravated by deep breathing and lying on the right side. He had suffered from a mild upper respiratory infection for one week prior to the onset of acute symptoms. He was admitted to a local station hospital where his temperature was 100.6° F. Physical examination was negative except for dullness, decreased breath sounds and râles at the right lung base. A chest roentgenogram (Fig. 1) revealed a large area of radiolucency at the right base with two fluid levels. On the second hospital day he developed a cough productive of one-half cup of greenish-yellow sputum. Antibiotic treatment was instituted and symptomatic improvement was noted in forty-eight hours. He became afebrile in two weeks and was transferred to Fitzsimons Army Hospital.

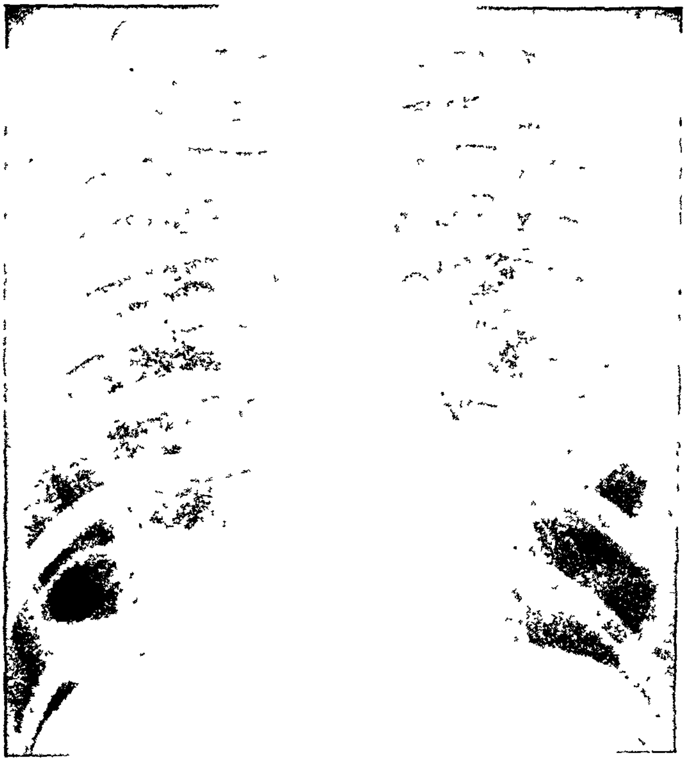


Fig. 1.—Posteroanterior roentgenogram of the chest The enormous cyst with two *fluid levels* is demonstrated on the right.

The past history revealed that he had had bilateral pneumonia in 1947.

The physical examination revealed a well-developed, well-nourished white man who did not appear acutely ill. The temperature, pulse rate, and blood pressure were normal. The only positive physical findings were confined to the chest which revealed *diminished* breath sounds, dullness to percussion, and râles at the right base, posteriorly.

The laboratory studies were all within normal limits.

A chest roentgenogram revealed two cysts in the posterobasal and superior segmental areas of the right lower lobe with fluid levels.

At operation, the basal segments of the right lower lobe were replaced by cystic changes. The entire lower lobe appeared grossly free of anthracotic pigment. In the

Additional laboratory studies, which included urinalysis, serology, and hemogram, were negative. All of the skin tests, tuberculin (PPD), histoplasmin and coccidioidin, were negative. A chest roentgenogram revealed a sharply demarcated lobular area of increased density which was quite homogeneous in the right lung field, located in the region of the cardiophrenic angle (Fig. 4). Roentgenograms of chest obtained prior to induction into the military service were reviewed and this lesion had been present since that time without change. A bronchogram demonstrated incomplete filling of the posterior basal segmental bronchi.

An exploratory thoracotomy was performed on Feb. 8, 1954, and a large cystic mass was palpated in the posterolateral and posterior basal segments. An anomalous artery measuring 8 mm. in diameter and 4 cm. in length was found in the free edge of the inferior pulmonary ligament. Its origin was from the aorta. The anomalous artery entered the area of the cystic change in the lower lobe. A right lower lobectomy was performed. There were no postoperative complications.

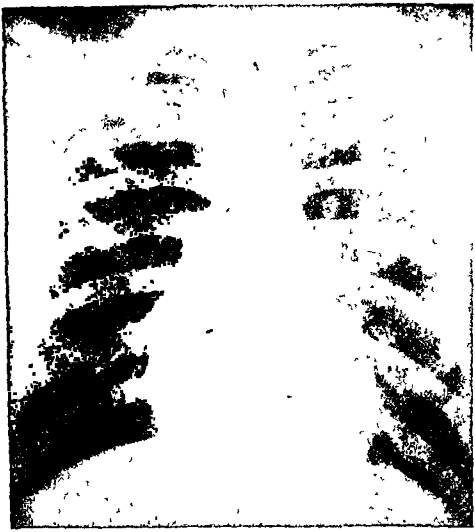
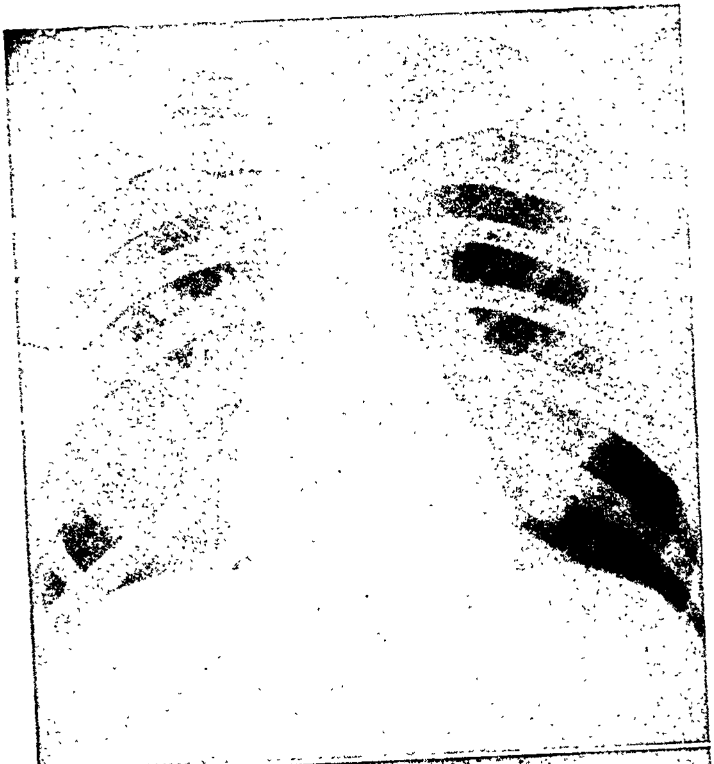


Fig. 4.—An area of increased density, fairly well demarcated, is visualized near the right cardiophrenic angle.

Gross pathologic examination revealed a cystic mass which measured 11 by 7 by 5 cm. in the area of the posterior and lateral basal segments. The cyst contained a grayish-brown grumous material which resembled an old blood clot. There was no bronchial communication between the cystic area and the normal bronchial tree. A large artery which measured 8 mm. in diameter entered the cystic mass and gave off numerous ramifications into the substance of the involved segment and about the walls of the cyst. One of the branches of this abnormal artery converged upward toward the hilum and anastomosed with a branch of the pulmonary artery.

Microscopic examination demonstrated the cyst to be lined by low columnar and cuboidal ciliated epithelium. The pulmonary parenchyma showed a mild degree of chronic organizing pneumonitis with mild inflammatory changes. A section of the large aberrant artery showed that it was of the elastic type. There were no atheromatous deposits found on the intima.

Fig. 2.



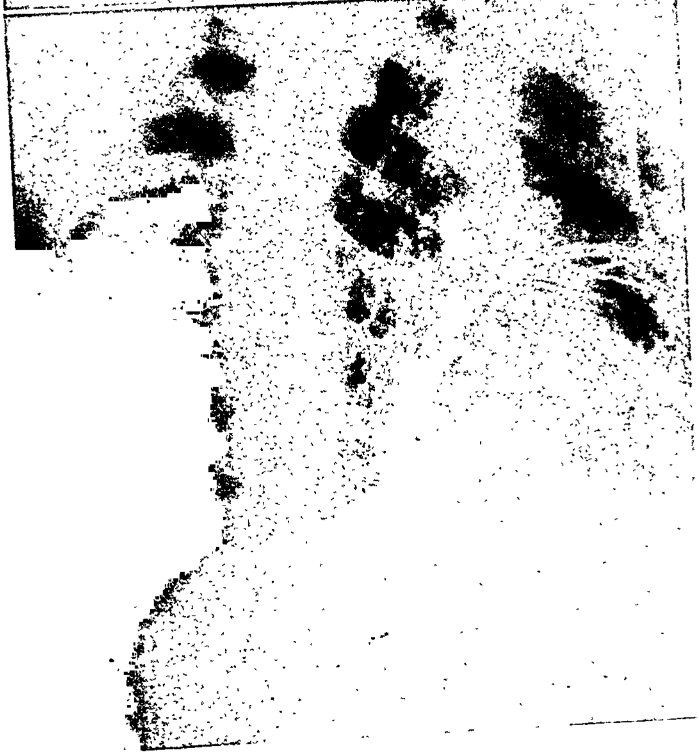


Fig. 3.

Fig. 2.—Posteroanterior roentgenogram of the chest. A rounded mass partially hidden by the left tenth rib is seen; increased radiolucency is noted below and lateral to the area of homogeneous density.

Fig. 3.—Right anterior oblique bronchographic view demonstrates a large emphysematous area not communicating with the bronchial tree and displacing all basilar bronchi. The dense mass is not shown on this view.

CASE 4—A 23 year old white man was transferred to Fitzsimons Army Hospital on June 6, 1955, for evaluation of an abnormal chest roentgenogram which was obtained in May, 1955

The patient had been hospitalized elsewhere for treatment of a peptic ulcer A chest x ray obtained in May, 1955, revealed an ill defined infiltration in the lower portion of the right lung field

The past history was completely negative

A complete physical examination was within normal limits

Laboratory examinations which included a complete blood count, urinalysis, and serology were normal A culture of the sputa for predominating organisms revealed alpha and beta hemolytic streptococci and *Neisseria catarrhalis* The tuberculin skin test (PPD), intermediate strength, was positive The histoplasmin and coccidioidin skin tests in a dilution of 1 100 were negative A bronchoscopic examination showed mucopurulent tenacious secretions coming from the orifice of the right lower lobe A chest roentgenogram (Fig 5) made in June, 1955, demonstrated a homogeneous density in the medial portion of the right lower lung field with a sharp superior and lateral margin but an indistinct inferior and medial margin Laminagrams revealed a loubulated density with clear cut margins in the right cardiophrenic angle which was thought to have a stringlike density extending downward and medially toward the diaphragm A lateral laminagram revealed that this mass lay in the posterobasal segment (Fig 6) An angiocardiogram was normal Anterior displacement of the posterior basal segmental bronchi was demonstrated by bronchography By retrograde aortography, a vessel was seen to arise from the right side of the aorta and extend up into the base of the right lung, supplying the area of increased density in the medial and posterior portion of the right lung field

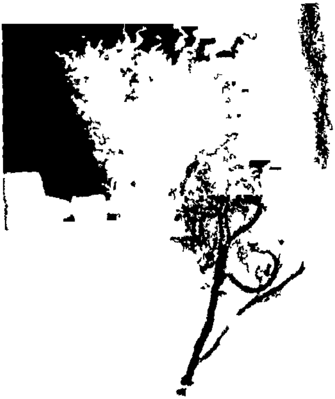
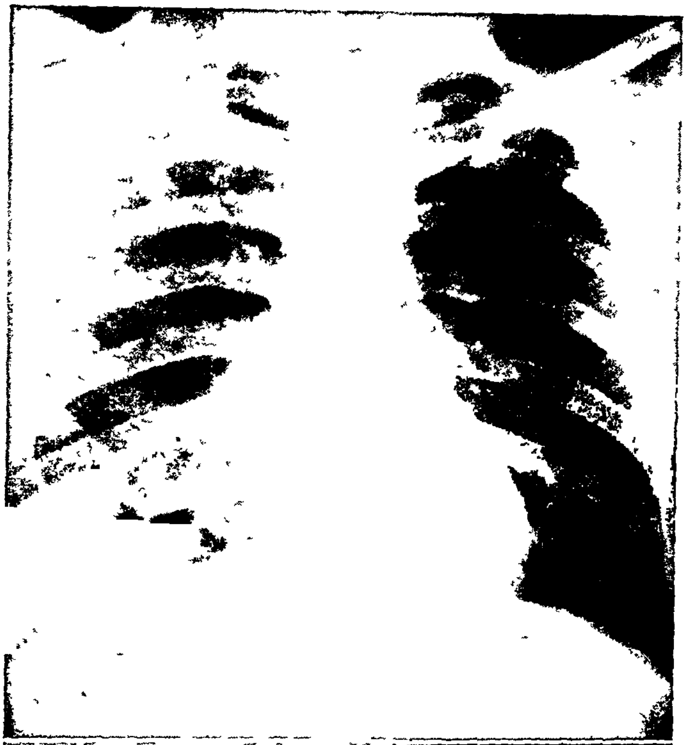


Fig 7—Roentgenogram of surgical specimen The aberrant artery is incompletely filled with Lipiodol The basilar bronchi are filled with the same contrast medium and attached to syringe

On Aug 8, 1955, an exploratory thoracotomy was performed The posterior one third of the right lower lobe was light pink in color There was no anthracotic pigment visible Palpation of this area revealed the presence of a comma shaped, rubbery, fluctuant density which measured approximately 5 by 4 by 3 cm An aberrant artery was noted in the inferior margin of the pulmonary ligament which measured 6 mm in diameter and originated from the aorta at the level of the right leaf of the diaphragm This artery measured 3 5

Fig. 5.



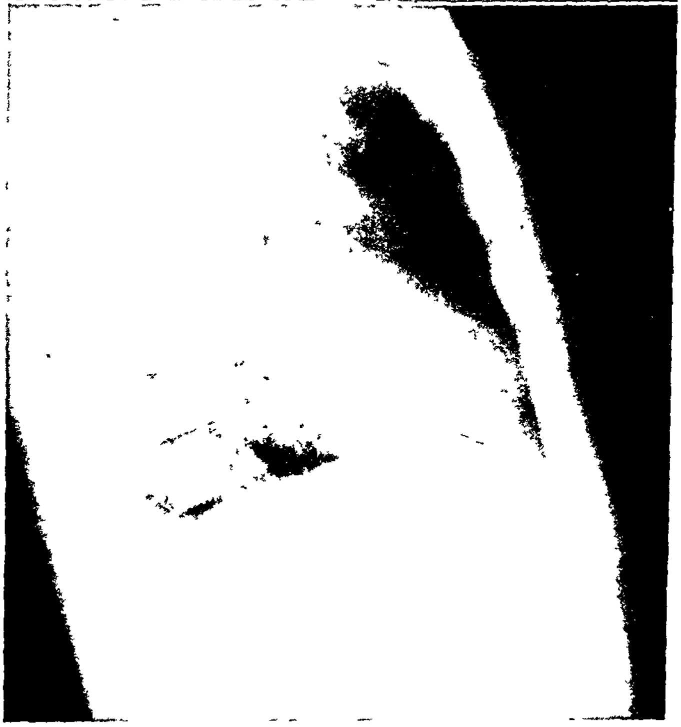
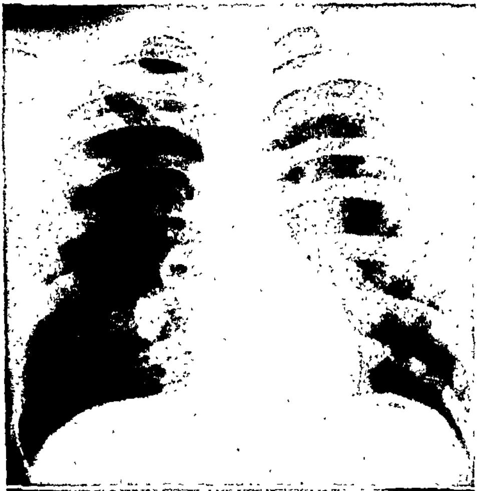


Fig. 6.

Fig. 5.—Roentgenogram demonstrates irregularly outlined area of density in the medial portion of the right lower lung field.

Fig. 6.—Circumscribed area of density localized to the area of the posterior basilar segment on lateral laminagraphic view.

Fig. 8.



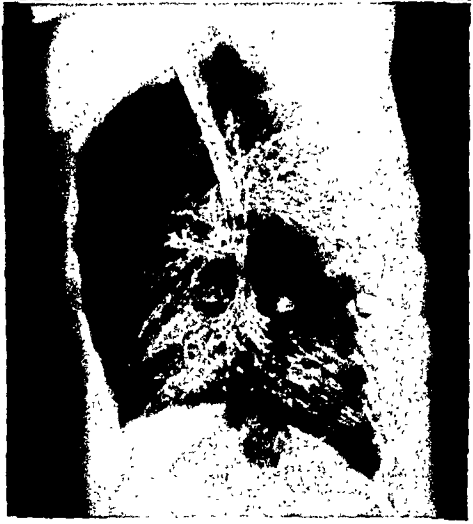


Fig. 9.

Fig. 8.—Roentgenogram of the chest. Area of density clearly visualized in the medial portion of the right lower lung field.

Fig. 9.—Lateral bronchographic view. Nonfilling in the region of the superior segment of the right lower lobe; localized density again viewed in this area.

cm. in length and entered the substance of the lung posteriorly in the region of the posterobasal segment. Resection of the basilar segments of the right lower lobe was performed without complication.

On gross pathologic examination, in the region of the posterobasal segment, a fluctuant mass was palpated which measured 4 by 3 by 2.7 cm. Approximately 20 c.c. of a creamy, tenacious, semisolid material was found within the cystic mass. An anomalous artery was found to enter the posterior and medial basal segments, which was approximately 5 mm. in diameter and seemed to encircle the cystic area in the posterobasal segment. There was no bronchial communication demonstrable (Fig. 7).

Microscopic examination revealed that the cyst was lined by ciliated columnar epithelium. In some areas cartilaginous plates were found. Within the pulmonary parenchyma a moderate amount of fibrosis and inflammatory reaction was noted. Sections through the anomalous artery revealed that there was an abundance of elastic tissue within the media. Atheromatous plaques were found in the intima in many sections.

CASE 5.—A 22-year-old white enlisted man was admitted to Fitzsimons Army Hospital on Oct. 10, 1955, for evaluation of an infiltrate in the lung of undetermined etiology.

In September, 1954, during basic training this patient suddenly developed a fever and cough productive of small amounts of purulent sputum. He was hospitalized at the local station hospital and treated for a period of two weeks. He recovered completely from this episode and was symptom free until August, 1955, at which time he noted the onset of ease of fatigue and a 15-pound weight loss which occurred over a period of one month.

The past medical history was completely negative.

The physical examination was within normal limits.

Laboratory studies revealed that the blood count, sedimentation rate, urinalysis, serology, and all of the skin tests to be negative. Gastric washings and culture for acid-fast bacilli were negative.

Chest roentgenograms made in September, 1954, revealed an increased density in the medial portion of the right lower lung field. Chest x-rays taken at Fitzsimons Army Hospital demonstrated an oblongated mass in the right lower lung field on posteroanterior view, which was localized to the posterobasal segment of the right lower lobe on lateral chest roentgenograms (Fig. 8). There was some suggestion of a cystic area peripheral to the homogeneous mass. Posteroanterior and lateral laminagrams showed that this was a "comma-shaped" density and that there was a suggestion of a vessel in the medial and inferior portion of this mass which extended downward toward the diaphragm. Peripheral to the homogeneous mass, an emphysematous area was noted which had the appearance of a pulmonary cyst. An angiocardiogram was within normal limits. Complete filling of all segments of the right lung except the superior segmental bronchus was demonstrated by bronchography (Fig. 9). A large cystic area occupied the region of the superior segment. A retrograde aortogram did not reveal any abnormal vessels entering the lung.

On Dec. 6, 1955, an exploratory thoracotomy was performed. In the region of the posterobasal segment the lung appeared quite pink and was without evidence of anthracotic pigmentation. A cyst which measured 5 by 4 by 3 cm. was palpated in this area. Just superior to the inferior pulmonary vein and along the course of the lower lobe bronchus, a large bronchial artery measuring 2.5 mm. in diameter was found which came directly up through the mediastinum from the aorta. This large systemic artery was observed to enter the substance of the emphysematous tissue and the cystic area in the posterobasal segment. Resection of the palpable, irregular cyst along with the attached posterior basal segment and the emphysematous parenchyma in the anatomic location of the superior segment was performed without undue difficulty.

On gross pathologic examination, numerous intercommunicating cystic structures varying in size from 1 to 4.5 cm. were observed in the region of the posterobasal segment. There was no bronchial communication to these cystic areas. A large anomalous artery measuring 3 mm. in diameter was found to enter the region of the posterobasal segment. There were two small branches of the pulmonary artery which entered the abnormal sequestered pulmonary tissue in the superior segment.

thoracotomy was performed on Dec 7, 1955, and an irregular cystic mass was found in the region of the inferior pulmonary ligament and was largely located in the lower posterior mediastinum. Posteriorly, below the level of the inferior pulmonary vein, a large systemic artery coming directly off the aorta was noted. The tense cyst was excised from the mediastinal structures where it was markedly adherent to the esophagus and the inferior vena cava. The patient's postoperative course was uneventful.

Upon gross pathologic examination, an anomalous artery was seen to enter the pulmonary tissue between the posterior and medial basal segments. A firm, irregular mass measuring 5 cm. in diameter was palpated in the mid portion of the basilar segments. The pulmonary tissue in the medial basilar segment and the medial portions of both the anterior and posterior basal segments had a bright yellow, granular appearance.

Microscopic examination revealed that the cyst wall was lined with bronchial epithelium. Surrounding the cyst, no pulmonary parenchyma, recognized as such, was present. A marked inflammatory infiltration was found in most sections. This reaction consisted of lymphocytes, plasma cells and, in some areas, polymorphonuclear leukocytes. A large number of macrophages containing fat droplets were observed in the medial basal segment. Most of these macrophages were within the alveolar walls. In the lateral basal segment, more normal appearing pulmonary tissue was seen. There were a number of these lipoid filled macrophages in this area also, but there seemed to be a sharp line of demarcation between the more solid lipoid filled areas and the relatively normal lung tissue.

## DISCUSSION

Intralobar bronchopulmonary sequestration as demonstrated in these 6 cases presents a rather typical roentgenographic picture, but no characteristic clinical signs and symptoms. Only one of our patients was ill during the period of hospitalization, but 2 had given histories of previous pneumonia. Whether or not these were related to the sequestered areas of the lung cannot be definitely stated. We know by review of previous chest roentgenograms on 2 patients that the lesion had been present prior to induction into the military service. Both these patients' lesions had been unchanged for approximately two years prior to entry into the military service.

It is interesting to note that laminagraphic studies on Cases 3 and 4 reveal practically identical x-ray pictures. The lesion was "comma shaped" in both instances and fingerlike projections were seen to extend from the inferior portion of the mass toward the diaphragm. The laminagram on Case 4 did not demonstrate this type of abnormality, however, it is our belief that due to mediastinal structures surrounding this cystic area, laminagraphic detail was obscured.

We do not feel that retrograde aortography is indicated when intralobular bronchopulmonary sequestration is suspected due to the possible untoward sequelae of this procedure. However, if an arteriovenous fistula of the lung is seriously considered, an angiocardiogram is indicated.

Although the true incidence of this condition is not known, we believe it to be more common than is ordinarily suspected.

The gross abnormalities of these 6 cases were uniformly similar, disregarding secondary inflammatory changes, and conformed to the pathologic pattern originally described by Pryce.[7] There was no bronchial communication demonstrated in any of our cases. The pulmonary parenchyma in the sequest

Microscopic examination revealed that these cysts were lined with bronchial-type epithelium. The pulmonary parenchyma presented evidence of pulmonary fibrosis and inflammatory changes.

CASE 6.—A 17-year-old Negro airman was transferred to Fitzsimons Army Hospital in October, 1955, because of an abnormal routine chest roentgenogram.

The patient had been completely asymptomatic until Aug. 1, 1955, at which time he developed a mild cough which was productive of a small quantity of thick sputum. There was no history of hemoptysis, nor was there any history of pulmonary infections in the past.

The past medical history was negative.

Physical examination revealed a well-developed, well-nourished Negro man who did not appear acutely or chronically ill. A complete physical examination was within normal limits.

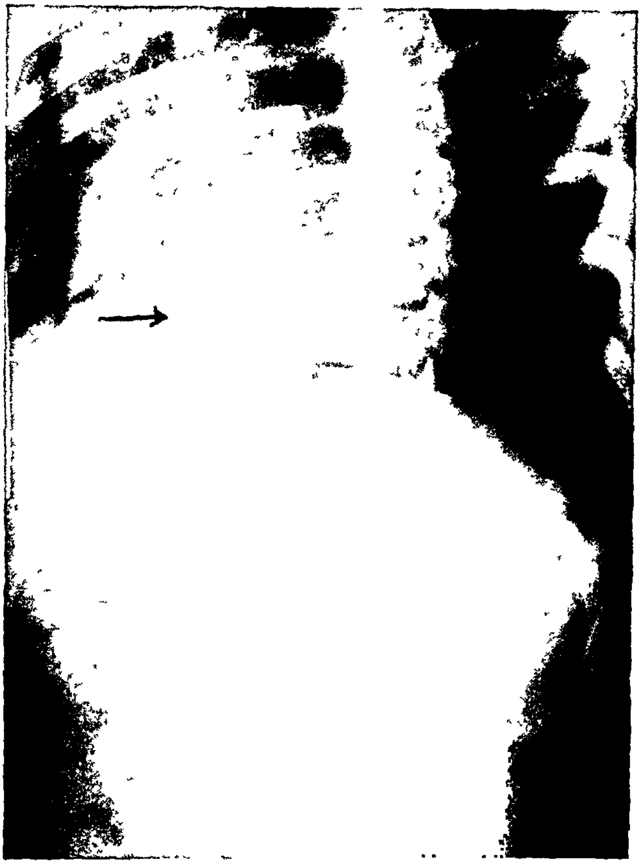


Fig 10.—Left anterior oblique roentgenogram Arrow points to the large circumscribed homogeneous density.

The laboratory examinations revealed a normal urinalysis, blood count, and serology. The tuberculin test (PPD) was positive. The histoplasmin skin test, 1:1000 dilution, was positive.

Additional chest roentgenograms revealed an area of increased density in the right cardiophrenic angle. An upper gastrointestinal series and barium enema were within normal limits. A bronchogram was normal. Posteroanterior laminagrams demonstrated an ill-defined abnormal shadow in the right lower lung field just posterior to the right cardiophrenic angle. Lateral laminagrams and left anterior oblique views revealed a rather sharply circumscribed, rounded cystic shadow which appeared continuous with the cardiac border and was seen best on the 11 cm. through 14 cm. sections (Fig. 10). An exploratory

# TRANSPLANTATION OF THE DIAPHRAGM FOR OBLITERATION OF DEAD SPACE FOLLOWING PULMONARY RESECTION

WALTER J. BURDETTE, PH.D., M.D.
COLUMBIA, MO.

THORACOPLASTY is the usual method of reducing the dead space following pulmonary resection, diminishing the amount of overdistention and loss of pulmonary function of the contralateral lung and of any residual homolateral segments remaining after partial resection. Although thoracoplasty does not result in appreciable external deformity when the first rib and transverse processes are preserved, we have found in practice that it is not performed routinely at the time of the primary procedure in the interest of reducing mortality and morbidity when the resection has been difficult and prolonged. When thoracoplasty is a secondary procedure, irreversible compensatory changes have usually occurred in the lung and the advantages of immediate reduction in volume of the thorax are lost. Thoracoplasty is not suitable when inferior segments are resected and may well result in bronchial torsion and impeded drainage of upper segments. Crushing the phrenic nerve for the purpose of elevating the diaphragm after lobectomy has the undesirable effect of reducing the respiratory excursion of the portion of the lung remaining, and plombage has been unsatisfactory except as a temporary measure in our hands. After suitable studies on dogs, one solution to this problem appeared to be superior transplantation of the diaphragm. A case in which the idea proved useful was that of a minister's wife, who was found to have an adenocarcinoma of the left lower lobe. This was resected, and the circumferential origin of the diaphragm was detached and sutured higher within the chest. Dead space was thus obliterated, and the patient was left with a functioning diaphragm immediately beneath the upper lobe which was not overdistended.

## CASE REPORT

Following six attacks of pneumonitis over a period of one year, the patient, a housewife 68 years of age, was hospitalized in a neighboring city with fever of 101° F. and a productive cough. She was treated with penicillin and streptomycin, but the fever persisted and roentgenograms showed a left pleural effusion and a shadow at the left lateral border of the heart. This was originally interpreted to be an aneurysm of the descending aorta but finally was diagnosed as an atelectatic lower lobe. She was discharged to her home but continued to have fever which usually was not higher than 99.5° F. Also dullness to percussion, distant breath sounds, and râles persisted over the left lower chest. Postural drainage yielded a small amount of blood-streaked sputum. Up to this time the patient had lost approximately 20 pounds.

---

From the Department of Surgery, The University of Missouri School of Medicine, Columbia, Mo.

Received for publication June 5, 1956.

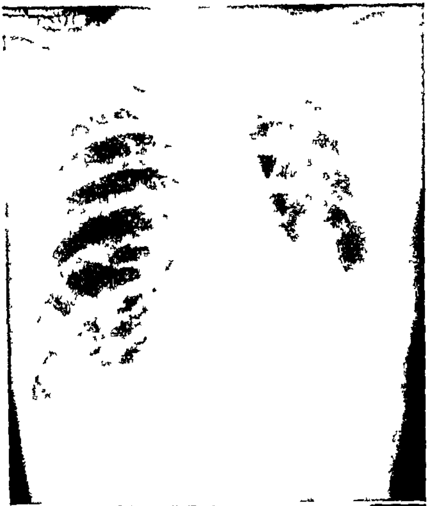
area was described as being light pink and visibly devoid of anthracotic pigment by each of the operating surgeons. However, microscopically, anthracotic pigment was occasionally demonstrated in these areas. The pulmonary tissue between and among the cystic areas showed evidence of fibrosis and inflammatory reaction. The anomalous arteries which were stained for elastic tissue in all cases revealed that there was an abundance of elastic tissue, thus, these anomalous vessels are designated as pulmonary type arteries. Intimal sclerosis was noted in one of the anomalous vessels.

The roentgenographic appearance of 5 of these 6 cases was almost identical in location and size. Although this is not diagnostic we feel that one's index of suspicion should be high if an abnormality is encountered in this area. As was stated previously, the clinical picture may be one of repeated respiratory infections or may be completely negative. Familiarity with this condition has enabled the diagnosis to be made in 5 of our 6 reported cases.

## REFERENCES

1. Bruwer, M. B., Clagett, O. T., and McDonald, J. R.: Intralobar Bronchopulmonary Sequestration, Am. J. Roentgenol. 71: 751-761, 1954.
2. Pryce, D. M.: Lower Accessory Pulmonary Artery With Intralobar Sequestration of Lung, J. Path. & Bact. 58: 457-467, 1946.
3. Pryce, D. M., Sellors, T. H., and Blair, L. G.: Quoted from, Intralobar Sequestration of Lung Associated With an Abnormal Pulmonary Artery, Brit. J. Surg. 35: 18-29, 1947.
4. Wyman, S. M., and Eyler, W. R.: Anomalous Pulmonary Artery From the Aorta Associated With Intrapulmonary Cysts (Intralobar Sequestration of Lung); Its Roentgenologic Recognition and Clinical Significance, Radiology 59: 658-667, 1952.
5. Boyd, G.: Intralobar Pulmonary Sequestration, Dis. Chest 24: 162-171, 1953.
6. Cole, F. H., Alley, F. H., and Jones, R. S.: Aberrant Systemic Arteries to the Lower Lung, Surg., Gynec. & Obst. 93: 589-596, 1951.

Fig 3



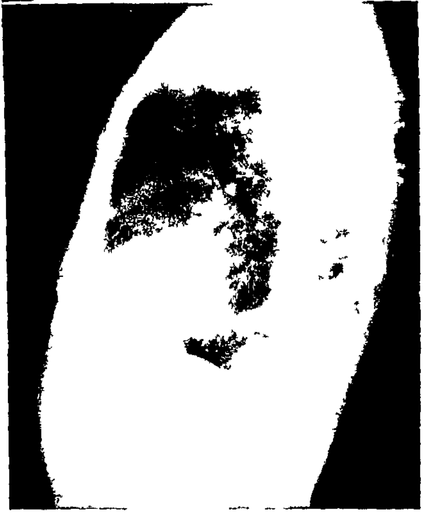


Fig 4

Figs 3 and 4 —Appearance of chest five weeks later with resorption of fluid and elevation of left leaf of the functioning diaphragm Space occupied by the eighth rib before operation is the level to which periphery of the diaphragm was transplanted

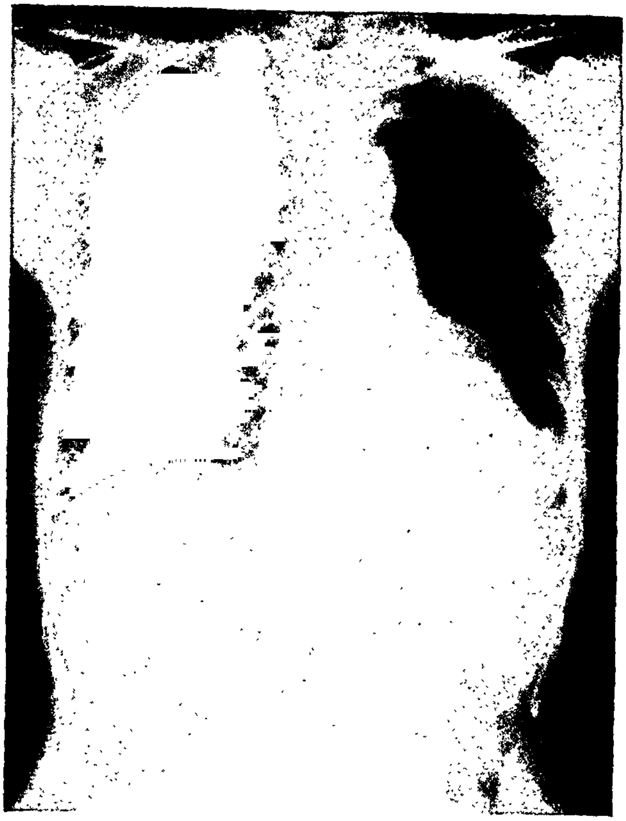


Fig. 1.—Roentgenogram of patient on admission with atelectatic left lower lobe containing adenocarcinoma.

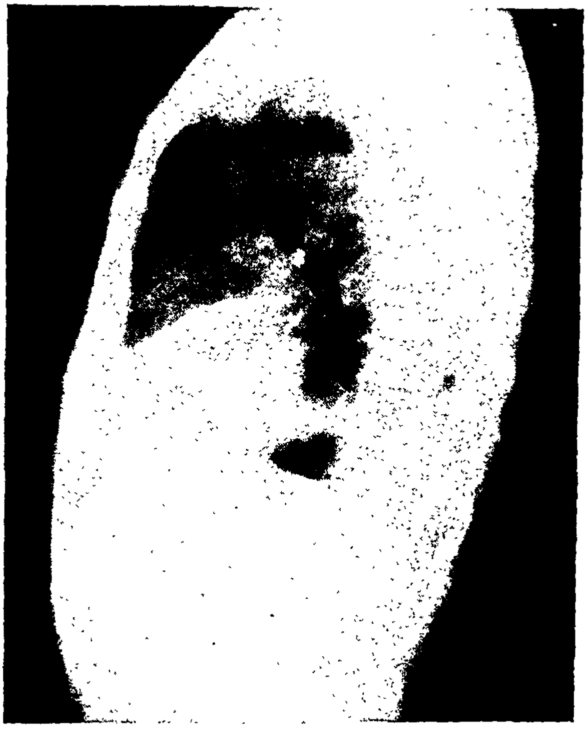


Fig. 2.—Lateral view following lobectomy showing accumulation of fluid beneath upper lobe and air in stomach and colon beneath elevated diaphragm.

# AUTOTRANSPLANTATION OF SPLEEN INTO THORAX

Edward F. Skinner, M.D.,* and Wm. W. Hurteau, M.D.**
Memphis, Tenn.

Experimentally, it has been shown that splenic tissue can be transplanted to other parts of the body and that such tissue will eventually grow in size. In the transplants, the red pulp usually predominates over the white pulp. The following case report is sufficiently unusual that we feel it is worthy of reporting. It is also interesting that this tumor could not be differentiated from a bronchogenic carcinoma until the time of surgical extirpation.

## CASE REPORT

Mr. R. H. was a 29 year old white man.

The patient stated that he had sustained a chest injury in 1945 (shrapnel wound) during World War II. At that time he had had an emergency operation and was informed that the spleen had been removed and a hole in the diaphragm had been stitched. He thought there was also some damage to the kidney. Since that time he had had periodic check up examinations with chest films made at six month intervals. A film made early in 1955 at the Board of Health in his community was reported as showing a "spot" in the left upper lobe. The patient was a retail lumber dealer, self employed, working ten to twelve hours daily. He had had chronic sinusitis up until a few years previously; otherwise he considered himself essentially well. He did not smoke.

*Past History*.—The patient gave a history of having had pneumonia at the age of three months and a tonsillectomy in 1930.

Physical examination was essentially normal except for the presence of a healed left thoracotomy scar. The blood pressure was 120/80 mm. Hg, a complete blood count was normal with a normal blood sedimentation rate of 9 mm. in the first hour. Numerous x rays were made of the chest and rib cage and yielded little further information except for the fact there was a spherical density, approximately 2.5 cm. in diameter, below the fourth left rib in the posterior axillary line. The outline of the mass was smooth and homogeneous. We felt it was probably in the lung, but also might involve the rib.

Previous x ray films made in 1951 were obtained and these revealed no large tumor on the left, but there was a very faint suggestion of a beginning mass in this same area over the left third rib, though not definite. On reviewing the series of films we came to the conclusion that the lesion had been present for at least eight or nine months without much change in 1955 and *if* it was present in 1951, it was certainly very small at that time.

Preoperative diagnoses included: (1) bronchogenic carcinoma, (2) hamartoma, (3) traumatic arteriovenous aneurysm, and some of the granulomatous diseases. Surgical removal was recommended in view of the uncertainty of the diagnosis.

*Operation*.—On Oct. 25, 1955, a left exploratory thoracotomy was performed through a standard posterolateral approach and multiple tumors were seen on the parietal and visceral pleura, the largest being over the third and fourth ribs, posteriorly. These tumors

---

Received for publication June 11, 1956.

*University of Tennessee College of Medicine Department of Surgery, Memphis, Tenn.
**St. Joseph's Hospital Department of Pathology, Memphis, T

Two months later, she was first referred to the University Hospital by Dr. J. E. Hill. On admission her blood pressure was 220/100 mm. Hg in the left arm and 218/98 mm. Hg in the right, the pulse was 78, respirations were 16 per minute, and the temperature was 99.8° F. The patient was thin, appeared to be chronically ill, and coughed frequently. However, the cough was only moderately productive. The heart was found to be enlarged 3 cm. beyond the midclavicular line; rhythm was regular, and apical systolic murmur of short duration was audible. Dullness at the left posterior and lateral base was found on percussion of the chest, and breath sounds were distant but only occasional râles were audible. A blood count showed 12 Gm. per cent hemoglobin and 14,500 white blood cells with 60 per cent polymorphonuclear leukocytes, 37 per cent lymphocytes, 1 per cent eosinophils, and 2 per cent basophils. A catheterized urine specimen was not abnormal, and an electrocardiogram showed normal sinus rhythm and no electrocardiographic evidence of cardiovascular disease. Sputum studies for neoplastic cells and pathogenic organisms were negative. Bronchoscopy revealed no intrinsic lesion, but thick mucoid secretions were found exuding from the left lower lobe, preventing adequate visualization. They were aspirated and the bronchoscope withdrawn. Following this, the patient was placed on postural drainage, and large amounts of sputum (up to 300 ml. per day) were expectorated. Although her febrile course improved, roentgenograms remained almost unchanged. A bronchogram showed failure of the segmental bronchi of the left lower lobe to fill, and operation was advised.

At thoracotomy, the lower lobe was found to be completely atelectatic and filled with mucoid material, and a lower lobectomy was easily performed since it was believed that the patient had insufficient pulmonary reserve to withstand pneumonectomy. After the fifth interspace was closed, the eighth rib was removed through the same skin incision, the periphery of the diaphragm was detached from the thoracic wall, and this free margin of the diaphragm was transplanted upward to the level of the eighth rib. The wound was then closed, leaving a fenestrated, No. 18 catheter for closed drainage of the thoracic cavity. Recovery was uneventful. Sections of the excised mass showed mucoid material throughout, and the pulmonary tissue present was compressed and reddish-brown in color. Proliferating papillary masses of epithelial cells, some of which contained mucin, were identified in sections, and a diagnosis of adenocarcinoma was made. No metastases were found in the lymph nodes removed. Other than occasional twinges of pain in the scar, the patient has had no respiratory symptoms following operation. Both cough and sputum have disappeared, and no dyspnea or digestive symptoms have been experienced subsequently. Hypertension has persisted (220/104 mm. Hg), but the patient now requires no medication and has completely resumed her normal activities three months later.

## SUMMARY

Transplantation of the diaphragm upward without disturbing the function of the phrenic nerve is useful in obliterating dead space and preserving function after pulmonary resection, particularly resection of inferior segments of the lung. A case in which this method was successfully used is reported.

## ADDENDUM

When the patient returned for her regular follow-up visit the following month, a small mass was found in the left breast. This proved to be adenocarcinoma with extension to axillary nodes, and a left radical mastectomy was performed. Pronounced divergence in the histologic appearance of neoplastic tissue from breast and lung favors the view that each represents a primary site but does not entirely exclude the possibility that the latter was metastatic from the former. Recovery was uneventful and without respiratory complications. Except for discomfort in the left upper quadrant of the abdomen with constipation, the patient has no complaints six months following the first operation.

had the general appearance of liver and were soft and smooth. The diagnosis of the pathologist at the operating room consultation was probable hemangio-endothelioma but, in view of the history of shrapnel wound, transplanted spleen was very possible; and paraffin sections would be required for definitive diagnosis. It was deemed advisable to excise the remaining lesions. The third and fourth ribs were removed en bloc together with the tumor attached to them, and the remaining four smaller tumors were removed by wedge excision of a portion of the lung and from the parietal pleura. Intercostal tube drainage was employed and the wound closed in the usual manner.

The patient's postoperative convalescence was essentially uneventful.

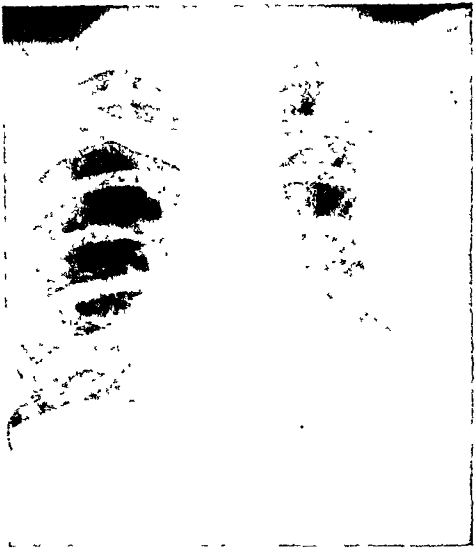


Fig 3.—Chest film made Nov. 2, 1955, one week following surgery. This film shows a defect in the left chest wall where the third and fourth ribs have been removed together with the tumor. There is some irregular pleural reaction noted on the left side which later subsided on thoracic exercises

*Surgical Pathology*—The specimen consisted of portions of two ribs, each of which measured approximately 14 cm. in length, together with intercostal muscle and attached fibrofatty tissue. The parietal pleura was present on the inner surface of this specimen. Attached to the parietal pleura at the center of the specimen was a tumor mass measuring approximately 3.5 by 2 by 1 cm. This tumor mass was covered by a smooth, gray, fibrous capsule and was ovoid in shape. On cut section, the fibrous capsule could be seen to penetrate into the tumor mass in the form of delicate fibrous trabeculae. The bulk of the mass was made up of a soft, pulpy, red, friable tissue which scraped readily with a knife. Very tiny, white, pin-point areas were dispersed throughout this tissue. The lesion was densely adherent to the parietal pleura (Fig. 4). An additional portion of tissue consisted of an oblong-shaped fragment of lung measuring 2.5 by 1.5 by 1 cm. On one surface of this was a 9 mm. portion of encapsulated tissue identical in all respects to that just described. A third portion of tissue representing parietal pleura measured 1 by 0.8 by 0.3 cm. This third portion of tissue again had an encapsulated 5 mm. nodule similar to that described

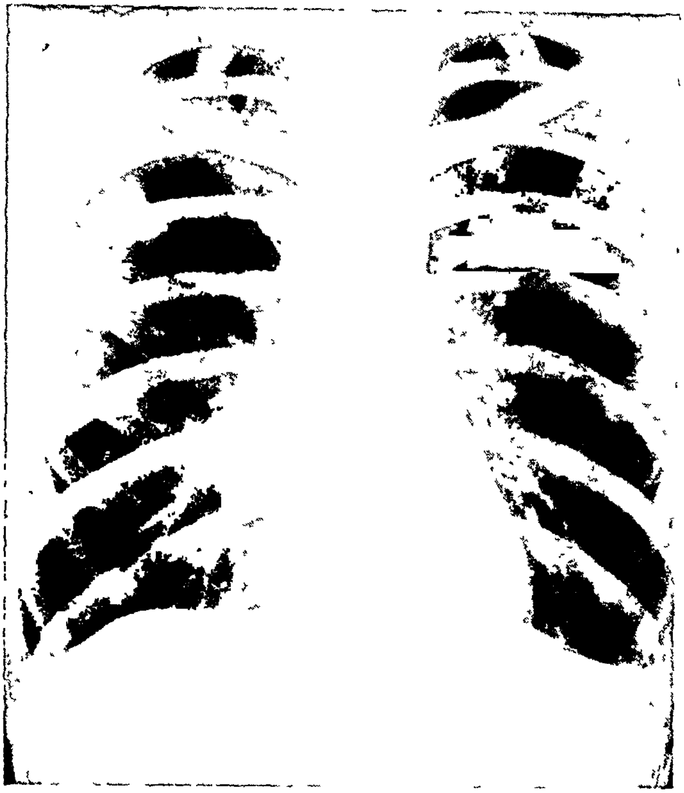


Fig 1.—Chest film made Feb 21, 1950, shows an essentially normal chest with no evidence of tumor.

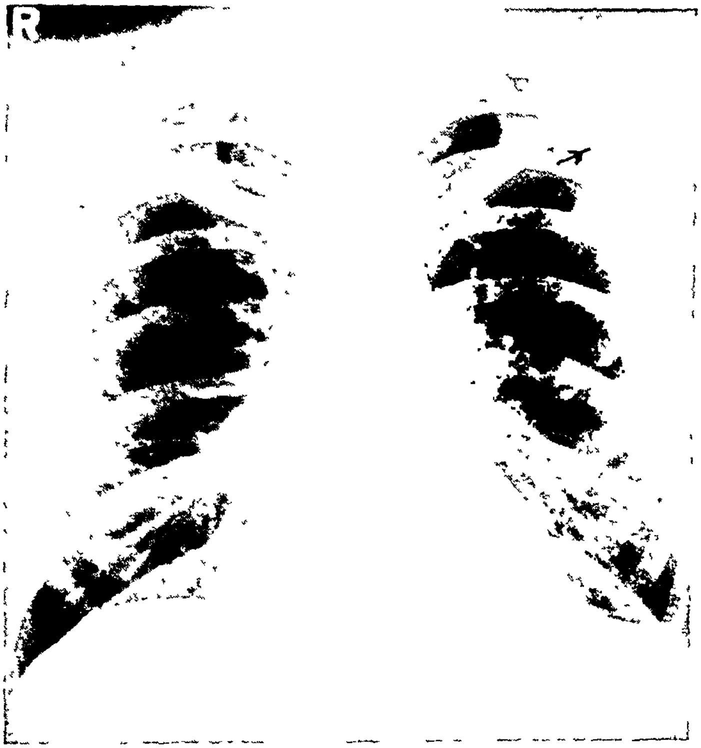


Fig. 2.—Chest film made Aug. 13, 1955. Tumor is now evident just below the fourth rib on the left posteriorly which seems to fuse with that rib. No other change is noted.

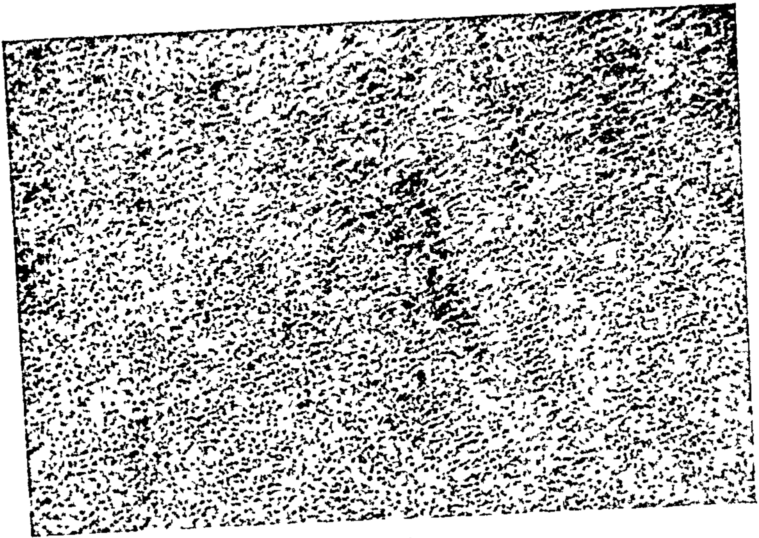


Fig 6

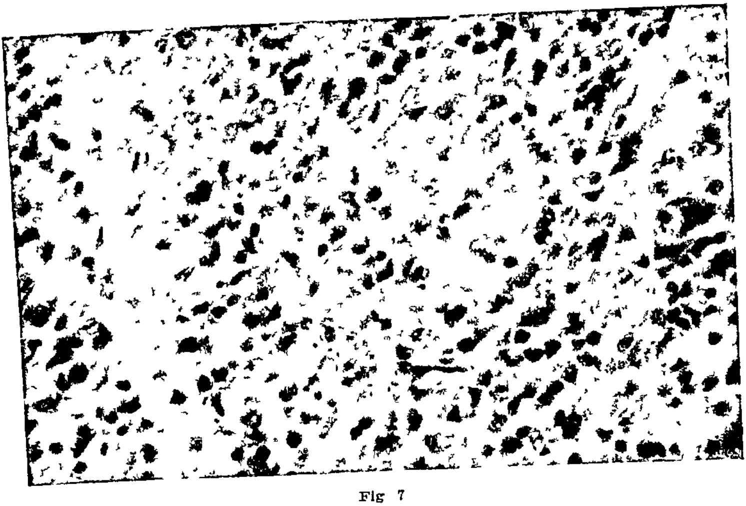


Fig 7

above. A fourth and a fifth portion of tissue, representing visceral pleura, measured 1.5 by 1 by 0.5 cm. These portions of tissue had attached encapsulated structures similar to that previously described, measuring 6 mm. in diameter.

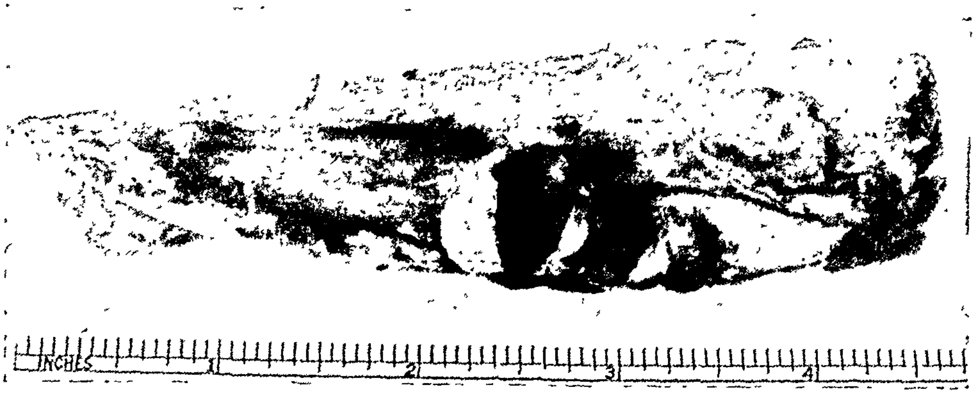
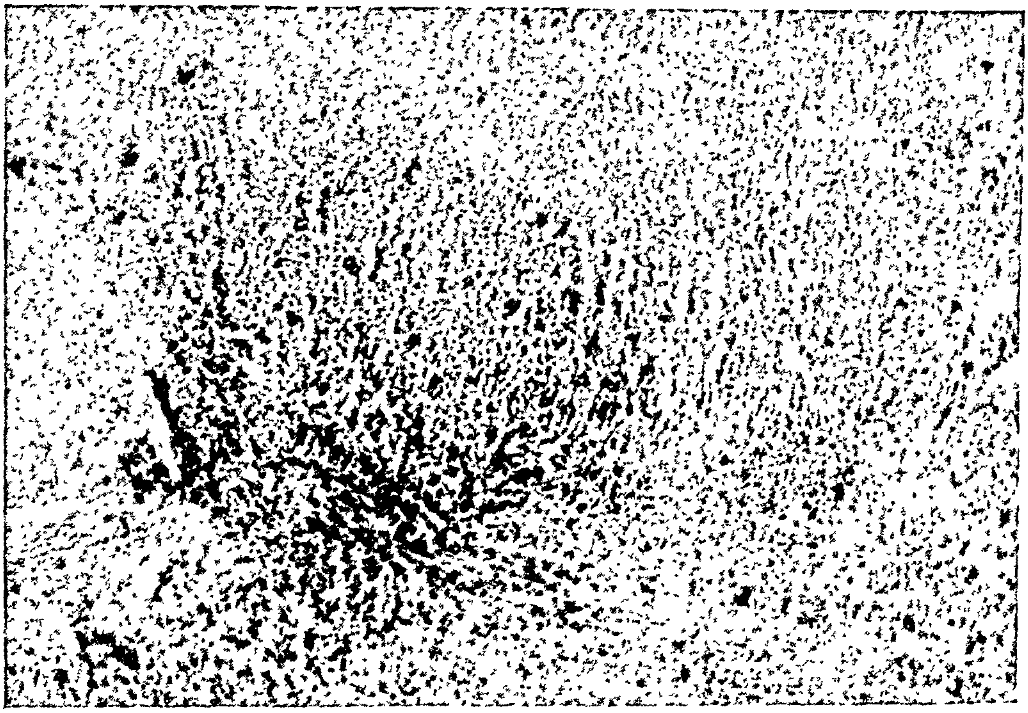


Fig. 4.—This is the gross specimen of the two resected ribs with the adherent tumor. The tumor has been split exposing its soft pulpy center.



Figs. 5-7.—Microscopic views of this tumor. Description is in the text.

*Histologic study:* All five portions of tissue showed an essentially similar picture except that the histologic details were best seen in the larger lesion attached to the ribs. These multiple lesions had well-defined connective tissue capsules. The connective tissue capsule in some areas was well vascularized and contained moderately large caliber arteries and veins. In the areas where the tumor came into relationship with the lung, the tumor was separated but adherent to the adjacent lung by a rather narrow zone of dense fibrous tissue. The pulmonary tissue showed dilatation of the alveoli with minimal focal areas of approximation of the alveolar walls. Macrophages, containing hemosiderin pigment, were present within the alveoli. The connective tissue capsule extended into the tumor in the

## SUMMARY

An unusual case is presented—the patient had a shrapnel wound of the left chest in 1945 at which time the spleen was ruptured, the kidney damaged, and perforation of the diaphragm occurred Apparently bits of the spleen were blown up into the chest The patient recovered from the original injury after an emergency operation and splenectomy He had no further trouble, until films were made in 1955 that revealed a tumor in the left upper chest In all, six splenic implants were removed from the visceral and parietal pleura and the lung tissue Microscopically, the bits of tissue represented well defined splenic tissue Although the patient was asymptomatic, surgery was recommended as bronchogenic carcinoma could not be excluded from the differential diagnosis It is of interest that in this case the red pulp of the autotransplanted splenic tissue predominated, similar to that seen in experimental transplants of splenic tissue

## REFERENCES

1 Jarcho, S and Anderson, D H Traumatic Autotransplantation of Splenic Tissue, Am J Path 15 527, 1939
2 Mylander, P A A, and Turrunen, M Transposition of the Spleen Into the Thoracic Cavity in Cases of Portal Hypertension, Ann Surg 142 954 956, 1955
3 Shaw, A F B, and Shafi, A Traumatic Autoplastic Transplantation of Splenic Tissue in Man With Observations on Late Results of Splenectomy in Six Cases, J Path & Bact 45 215 1937
4 Von Stubenrauch Milzregeneration und Milzersatz, Verhandl d deutsch Gesellsch f Chir 41 213 215, 1912
5 Von Stubenrauch Verlust und Regeneration der Milz beim Menschen, Beitr z klin Chir 118 285 305, 1920
6 Von Stubenrauch Experimental Untersuchungen uber die Entstehung der sogenannter Nebenmilzen, insbesondere nach Milzverletzungen, Beitr z klin Chir 119 710 714, 1920

form of connective tissue trabeculae. The vascular bed, likewise, continued into the tumor and collections of lymphocytes extended parallel to the vascular penetration. Aggregates of lymphocytes or lymphatic follicles were present which very much resembled the white pulp of the spleen. These nodules were made up of lymphocytes of various sizes supported by a delicate reticular framework (Fig. 5). Occasional follicles of this nature had lymphocytopoietic centers. These lymphatic follicles were relatively few in number as compared to the great majority of the tissue making up the bulk of the lesion which had the following appearance. There were multiple venous sinuses separated by cords of tissue. These venous sinuses were lined by rod-shaped cells arranged in a parallel manner in many areas while in other areas the lining cells were of a more compressed cuboidal appearance. These sinuses contained many erythrocytes, occasional macrophages, and occasional polymorphonuclear leukocytes. The sinuses were separated by cords of cells which ran in all directions. These cords of cells were made up of nongranular leukocytes, many lymphocytes, monocytes, and macrophages. The cords of cells were identical in appearance to the splenic or Billroth cords of the spleen and the sinuses were identical to the venous sinuses of the spleen (Figs. 6 and 7). These constituent parts of the red pulp greatly predominated over the white pulp.

## *DISCUSSION*

A survey of the literature reveals that cases of traumatic spread of splenic tissue to the peritoneum are quite rare.

Von Stubenrauch[4-6] considered that these splenic implants should be called "splenoids"; he thought that splenic autotransplants tend to regress, with the passage of time. His assumption that the peritoneum can manufacture splenic tissue is poorly supported.

Autoplastic transplantation of splenic tissue has been successful in the hands of many experimenters.

Such scattering of splenic tissue over the peritoneum following trauma is likely due to the dispersal of blood and bits of splenic pulp.

Jarcho and Anderson[1] add 2 new cases to the literature—both following trauma over the spleen. They summarize 12 other cases from the literature.

Mylander and Turrunen[2] report 3 cases of hypertension of the portal vein which were treated by transposition of the spleen into the pleural cavity in the hope that resulting adhesions and anastomoses would provide additional venous drainage. All 3 patients were in such poor condition that extensive surgical measures seemed out of the question. The results after a one- to two-year follow-up period suggested further studies concerning this method are well worth while.

We would be a little bit suspicious of this method in view of our one case of accidental transposition of the spleen into the thoracic cavity, as the spleen in our case continued to grow slowly over a period of many years. The difference in the case would be, of course, that in their case the spleen was transplanted without breaking its capsule, whereas in our case the spleen was blown into fragments and these segments were then distributed throughout the lung.

Fifteen years ago, Dr. Charles Olim and Dr. Duane Carr had a somewhat similar case at the John Gaston Hospital in Memphis. The patient had a ruptured spleen removed and several years later a rib tumor in this same area was excised and the rib itself contained splenic tissue growing within the marrow cavity. This patient also made an uneventful recovery.

clude not only candidates for surgery but also inoperable or postoperative patients transferred from other hospitals for radiation and palliative care. Thus, both early and terminal cases were seen.

The group being reported here is not a representative sample. In the first place, despite constant vigilance and effort, not all cases of lung cancer have been seen. This is inevitable in a large hospital with the patients scattered through several wards. Second, after they have been seen many of them leave the hospital and drop out of contact. Some die at home, some move away, some are still alive. A great majority of them, of course, do return for follow-up care, but in not all of these patients who die in the hospital is autopsy permitted. Thus, of more than 80 patients examined over the two-year period of the study, a total of 27, with complete autopsy, comprise the material for this analysis. A large proportion of these represent patients seen in the last three months of life.

The question that is posed of the two screening methods here being tested is simply whether or not metastases are present in the brain. The question of localization does not arise since these patients are not usually considered candidates for craniotomy. For this simple question the closer the autopsy confirmation follows upon the screening tests the better. So the fact that the majority of this group were terminal cases is favorable for purposes of this analysis.

The neurological examinations were performed by one individual (the writer) and the electroencephalograms were interpreted by him and also independently by Dr. Knox Finley, Neurology Consultant to the hospital. The electroencephalograms were all performed by the same technician on a Grass 8 channel machine using bipolar technique with routine electrode placement over the frontal, central, occipital, and temporal areas. Both interpretations were described separately and a definite statement made as to whether the record suggested lateralized or focal pathology.

## RESULTS

TABLE I. RESULTS

| BRAIN PATHOLOGY PREDICTED | | | BRAIN PATHOLOGY FOUND (AT AUTOPSY) | |
|---|---|---|---|---|
| | | | YES | NO |
| EEG<br>Clinical | Yes<br>Yes | 9 | 9 | 0 |
| EEG<br>Clinical | No<br>No | 11 | 1 | 10 |
| EEG<br>Clinical | Yes<br>No | 2 | 0 | 2 |
| EEG<br>Clinical | No<br>Yes | 5 | 5 | 0 |

These 27 cases may be divided into four categories:

I. Both clinical and electroencephalogram examination negative.
II. Both clinical and electroencephalogram examination positive.
III. Clinical examination positive and electroencephalogram negative.
IV. Clinical examination negative and electroencephalogram positive

# DETECTION OF INTRACRANIAL METASTASIS IN LUNG CANCER: A COMPARATIVE STUDY OF THE NEUROLOGICAL EXAMINATION AND THE ELECTROENCEPHALOGRAM

JAMES K. SMITH, M.D.
OAKLAND, CALIF.

## INTRODUCTION

THE high incidence of cerebral metastases in cases of bronchogenic carcinoma has been amply documented although the reports of incidence vary somewhat. Thus, early writers such as Adler[1] and Simpson[2] reported the percentage of cerebral metastases to be 14 per cent and 13.6 per cent, respectively, while more recently King and Ford[3] reported an incidence of 27 per cent and Halpert[4] gave a figure of 32.6 per cent, all studies being based on surveys of autopsy material.

Although the incidence of cerebral metastasis during life, in a group of patients with lung cancer at an earlier stage of the disease, will be considerably lower than the incidence at autopsy, it is still of sufficient frequency to justify a careful search in the individual case. This may be of considerable importance in the preoperative evaluation of a candidate for surgical removal of a pulmonary neoplasm.

In this hospital, the routine screening of such patients has included biopsy of prescalene lymph nodes and a radiologic survey of the skeletal system. These precautions are based upon the knowledge that bronchogenic carcinoma metastasizes very frequently to these locations. Yet up to two years ago, neurological screening of these patients was elective. In most cases, a neurological clearance was requested if the patient's physician felt suspicious but not otherwise. The method of screening also varied. Some physicians specifically requested a neurological consultation, others first asked for an electroencephalogram, being satisfied if this was negative.

The assumption implicit in this second procedure seems clearly to be that the electroencephalogram in itself is an effective screening technique for the presence of intracranial lesions. It was in large part to test the validity of this assumption that the present plan of study was instituted.

## METHOD

For the past two years, ward physicians throughout the hospital have been asked to refer all patients, with a diagnosis of lung tumor, for an electroencephalogram and a neurological examination. A skull x-ray was already included as part of the routine skeletal survey. Lumbar puncture was not performed routinely but only to verify suspicions arrived at by the clinical methods. In order to have as large a group as possible, it was decided to in-

---

From the Veterans Administration Hospital, Oakland, Calif.
Received for publication July 23, 1956.

these had cerebellar tumors In all of these cases, the localization was correctly made on clinical examination (Cases 15 16, 18) One patient presented also with a cerebellar syndrome clinically but at autopsy did not have metastatic deposits but rather showed degenerative changes throughout the cerebellar cortex with falling out of Purkinje cells (Case 19)

The fifth case, a right frontal lobe lesion was erroneously localized to the cerebellum on the basis of neurological findings (Case 17)

CASE 15 The patient was a 59 year old white man who had had a pneumonectomy in October, 1955, followed by x ray therapy He was discharged a month after surgery essentially asymptomatic and remained well until mid January, 1956 when he developed severe headache, felt dizzy, and became unsteady in gait Neurological examination on Jan 18, 1956, the day after admission, showed horizontal nystagmus on looking to the right, drifting of the outstretched left arm and slight terminal tremor on left finger to nose maneuver, and gross ataxia on placing left heel to right shin He was unwilling to walk for fear of falling The clinical impression recorded was one of left cerebellar metastasis The electroencephalogram performed Jan 24 1956, contained a mixture of basic 8 to 10 per second and occasional low voltage 4 to 6 per second cycles without focal or lateralizing pattern It was interpreted as normal to border line The patient died on Feb 6 1956

At autopsy, there was a solitary 2 by 3 cm tumor mass in the left cerebellar hemisphere

CASE 16 This 32 year old white man was admitted to the Neurological Service on Oct 17, 1955, because of severe pain radiating from the back of the neck into the right side of the head and behind the right eye The pain had commenced about two months before his entry to the hospital and had become incapacitating in the previous ten days On examination the chief finding was the pronounced guarding of the neck muscles, with a slight tilting of the head (often an early sign of cerebellar neoplasm) The spinal fluid, which was clear, was obtained under an initial pressure of 190 mm with a closing pressure of 100 mm It contained 2 cells 79 mg per cent protein, 2+ Pandy and the gold curve was 2234431000 Skull films were normal, and the chest film showed a nodular mass in the right hilum, "suggesting neoplasm" An electroencephalogram made one week after entry was entirely normal The patient went downhill despite an attempt at palliation with x ray therapy He developed evidence of further neurological deterioration with confusion and bilateral pyramidal tract involvement, and died on Nov 22, 1955

At autopsy he had an epidermoid carcinoma in the right lung and multiple metastatic nodules in both cerebral hemispheres as well as a large tumor mass occupying most of the right cerebellar hemisphere and two smaller ones in the left cerebellar hemisphere

CASE 17 This patient, a 63 year old white man, was examined on Feb 7, 1956, just three days before his death He had been receiving x ray therapy for an inoperable carcinoma of the right lung He had noted weakness of the left foot about one week previously He walked with steppage gait on the left and could not dorsiflex his left foot The left ankle jerk was less active than the right and there was diminished sensitivity to pinprick over the dorsolateral aspect of the left foot and the lower lateral calf The knee jerks were equal The reflexes in the left upper extremity were slightly brisker than those on the right The remainder of the examination was normal except for a rapid horizontal nystagmus, more marked on right lateral gaze The clinical impression was of metastases to the lumbosacral roots and to the cerebellum The electroencephalogram was normal

At autopsy, however, there was a solitary 2 cm rounded metastatic nodule in the central white matter of the right frontal lobe just anterior to the lateral ventricle The spinal cord and cauda equina were not examined

CASE 18 The patient, a 56 year old white man, was admitted to the hospital on March 2, 1955, for investigation of a persistent cough of three months' duration The

I. *Cases in which both clinical examination and electroencephalogram were negative:* There were 11 of these and the autopsy confirmed the ante-mortem impression in ten instances. The exception is summarized herewith.

CASE 1. The patient was a 66-year-old white man who was examined on Jan. 28, 1955, having had a pneumonectomy for small-cell carcinoma of the right lung in March, 1954. He appeared weak but had no complaints of neurological significance. He was mentally clear, walked with normal gait and showed no defects in coordination or strength of extremities. Cranial nerves were intact. The reflexes were equal. An occasional upward movement of the left great toe on the Chaddock maneuver was noted but plantar stimulation yielded a normal response. This single finding was not felt in itself of sufficient weight to make a diagnosis of metastasis. The electroencephalogram was normal. The patient was not seen again in neurological consultation but according to the progress notes of the ward physician it is clear that by the end of February he was becoming confused. He went home for a brief furlough in March and on returning to the hospital was lethargic and disoriented at intervals. He became stuporous terminally and died April 18, 1955, almost three months after the time of the neurological examination. At autopsy he had a large right temporal lobe tumor with fresh hemorrhage in its periphery.

*Comment:* It seems fairly certain that this patient had either not yet developed spread to the brain at the time of his neurological and electroencephalogram examinations or else (as seems likely) the tumor was then too small to have produced significant dysfunction or electrical abnormality.

II. *Cases in which both clinical examination and electroencephalogram were positive:* Of the 9 cases where both clinical examination and electroencephalogram correctly identified the presence of intracranial metastasis, there was one false localization.

CASE 2. The patient, a 54-year-old white man, entered the hospital Sept. 18, 1954, for terminal care of a carcinoma of the right lung. He had had an exploratory thoracotomy elsewhere three months earlier and an inoperable lesion was discovered. However, as early as three months before the thoracotomy, his wife reported that he had shown changes in his personal habits and she noted that he bumped into things occasionally as though he did not see them. When seen on September 27, two days before death, he was confused, disoriented, and yawned frequently. He could not cooperate well in the examination and was unable to walk, having been confined to bed ever since his chest surgery. There was no choking of the discs, no field defect, and ocular movements were normal. There were no striking abnormalities in the reflexes, motor strength, or sensation. It was the clinical impression that he had frontal lobe metastases. The skull films showed questionable erosion of the posterior clinoid process. The electroencephalogram showed bilateral frontal 4 to 6 per second waves of high amplitude, of greater prominence on the left. At autopsy he had a large tumor in the vermis of the cerebellum, extending into the vicinity of the dentate on each side. The brain was moderately swollen and there was some symmetrical ventricular enlargement, but no lesions were found in the cerebral hemispheres.

*Comment:* Here the terminal occurrence of edema with its consequent effect on psychologic function dominated and obscured the original lesion in the cerebellum. The electroencephalogram also falsely localized this lesion. The projection of delta waves into the frontal areas from posterior fossa lesions has been described by others.[7, 9]

III. *Cases in which the clinical examination was positive and the electroencephalogram negative:* There were 5 patients in this category. Three of outpatient clinic throughout their postoperative course as long as they remain in easy traveling distance. While some may prefer to spend their terminal stages at the family home, this is probably less likely to be true for those who develop cerebral metastases, since they often become difficult to care for at home. Hence, a greater relative proportion of this group return to the hospital and the autopsy statistics are correspondingly "loaded."

Of the 6 cases missed by the electroencephalogram, one was also missed on the neurological examination (Case 1) and, as has been pointed out, this is probably because the lesion was incipient when the patient was first seen. The other 5 were clear misses for the electroencephalogram, since the clinical opinion at the same time as the electroencephalogram correctly favored the presence of intracranial masses. Four of these were posterior fossa lesions. It has been stressed by various writers that this is a "blind spot" for the electroencephalogram.

Smith and associates[5] reported a pattern, which they felt was diagnostic of subtentorial tumor, consisting of delta waves over the occipital leads. Others have not found this to be a consistent pattern.

Rheinberger and Davidoff,[6] after reviewing a group of subtentorial tumors in adults, concluded that, "There is no pattern or distribution of electrical abnormality specifically indicative of posterior fossa disturbance."

Pupe and co-workers[7] pointed out that in the presence of posterior fossa tumors the electroencephalogram may point either to the midline or to the cerebral hemispheres.

Daly[8] reported a large series (66) of verified posterior fossa tumors studied by electroencephalogram and came to the conclusion that there was no predictable correlation between the site of the electroencephalographic abnormality and the anatomic site of the lesion. They found, as had Cuneo and his co-workers,[9] that the most commonly observed pattern was one of rhythmic bilaterally synchronous slow waves. Eighteen per cent of the cerebellar tumors in Daly's series presented normal electroencephalographic records.

The sixth case with a "false negative" electroencephalogram (Case 17) was a deep anterior frontal lobe nodule and the normal electroencephalogram only three days before death should serve to emphasize again that there are other silent areas for this test besides the posterior fossa. In this case, there was no significant edema of the brain on the side of the tumor and this, plus its deep location, probably accounts for the normal electrical activity from the overlying frontal cortex.

The embarrassing and familiar error of confusing tumor of the frontal lobe and the cerebellum was made twice on clinical grounds and in both possible directions (Cases 7 and 17). The electroencephalogram compounded the error in the first of these.

Finally, it may be of interest (though aside from the main purpose of the study) to point out that in the course of this screening, 4 patients were found to have evidence of brain metastases who would be otherwise considered operable for their lung tumor. The statistical significance of this figure (4 out sputum was positive for tubercle bacilli but bronchoscopic examination revealed an obstruction. Thoracotomy was performed, at which time an inoperable lung tumor on the right was discovered. The biopsy proved it to be adenocarcinoma. Neurological examination on July 1, 1955, indicated a left cerebellar lesion manifested by nystagmus in left horizontal gaze and past-pointing with left finger-to-nose maneuver. Skull plates were negative. The electroencephalogram made on July 8, 1955, was normal. Spinal fluid obtained on July 5, 1955, was under normal pressure and contained no cells, 72 mg. protein, 1+ Pandy and gold curve of 1112210000.

The electroencephalogram was repeated Sept. 8, 1955, at which time a right temporoparietal focus was present. The patient died Dec. 11, 1955, and the autopsy showed carcinoma of the upper lobe of the right lung, a large necrotic semicystic metastasis occupying about one half of the left cerebellar hemisphere and a 5 cm. lesion in the right parietal lobe.

CASE 19. This 61-year-old white patient was admitted to the hospital Oct. 18, 1955, for investigation of cough, weakness, and weight loss of three months' duration. On admission, he had physical signs and x-ray evidence of a mass in the right upper lung. He walked with a wide base, showed nystagmus on horizontal gaze, more marked to the left than the right, and performed the heel-to-shin maneuver with marked ataxia. The electroencephalogram was normal. Spinal fluid was under normal pressure and contained 3 lymphocytes, protein of 30 mg. per cent, negative Kolmer, and colloidal gold curve 0111100000.

He deteriorated steadily and died March 14, 1956. At autopsy, there was found an adenocarcinoma of the right lung. The brain was grossly normal, but the cerebellum showed diffuse falling out of Purkinje cells, the remaining Purkinje cells showing chronic degenerative changes.

*Comment:* The cerebellar degeneration in this patient probably falls into the group of carcinomatous degenerations of the central and peripheral nervous system which has recently been described in the literature.[10]

IV. *Cases in which the clinical examination was normal and the electroencephalogram abnormal:* There were 2 such cases. In both instances, the patients were terminal, cachectic, eating poorly, and requiring large doses of analgesic medication. The abnormality in the records was not sleep artefact but may have been attributable in part to metabolic and electrolyte disorders associated with the terminal phase of cancer. The records both showed bursts of diffuse 2 to 3 per second and some 5 to 7 per second slow waves and presented sufficient asymmetry to suggest lateralization of pathology to the two independent interpreters. The gross and histologic appearance of the brains was normal, however. In both these cases, the neurological opinion was that no metastatic brain lesions were present.

## COMMENT

Of the entire group of 27 autopsied cases, there were 15 with intracranial disease. Fourteen of these were detected by the neurological examination during life, while 9 of them had electroencephalographic abnormalities of focal type. It is apparent that the incidence of central nervous system metastases in this small group is preternaturally high—52 per cent in contrast to the 14 to 32 per cent range cited above.[1-4] The reason for this cannot be definitely stated, but some of the following factors may enter in. Hospitalization is readily available to veterans. These patients are followed closely in a surgical

# RIBBON ESOPHAGOSCOPES

ERNEST B EMERSON, JR M D
ROCHESTER, N Y

THE first ribbon esophagoscope was designed to solve a specific problem in handling an esophageal stricture in a patient after all other treatment had failed[1] After working with the instrument over a period of time, it soon became apparent that a tube of this shape had many advantages for the operator doing diagnostic and operative instrumentation

A set of two of these esophagoscopes is presented here in the belief that they will be a true addition to the armamentarium of the surgeons doing thoracic work

### THE INSTRUMENTS

The barrel of the new esophagoscope is a flat oval in shape to take advantage of the fact that the esophagus may be stretched from side to side more than it can in the anterior posterior diameter The extreme flattening of the barrel results in less laryngeal stimulation from pressure and hence less trouble with coughing and gagging during instrumentation The barrels are 51 5 cm and 29 5 cm in length The inside diameters are 9 by 19 mm

The light carrier is on the side of the barrel again taking advantage of the fact that the esophagus dilates more easily from side to side thus a full lumen is obtained It may be withdrawn and replaced by the operator if the light becomes clouded

At the proximal end of the esophagoscope there is incorporated a light trap, 6 6 cm in length This makes it possible to work in a brightly lighted room without interference with the operator's visual accommodation

The electric power supply is incorporated into the handle of the instrument, thus doing away with the proverbial nuisance of wires, and obtaining a constant power supply which will not burn out the light bulb in the midst of instrumentation

An advantage of the width of this barrel, especially to those men frequently dealing with carcinoma, is that the biopsy forcep may be passed diagonally from proximal to peripheral end of the scope so that the business end of the biopsy forceps is under direct observation when the specimen is obtained

---

From the Department of Surgery Division of Otolaryngology University of Rochester School of Medicine and Dentistry Rochester N Y
Received for publication July 5 1956

of 27) should probably be discounted, since it has been pointed out that the entire group was more heavily represented by terminal than by early stages of the disease. But it is worth repeating that in these 4 there was no other evidence of metastasis and the lung tumor itself appeared operable so that the patient was considered in all other respects an entirely suitable candidate for surgery. In 3 of these the neurological symptoms were prominent and could scarcely have been overlooked by any physician taking a routine history. In one, however, no specific neurological complaints were offered and the diagnosis was made upon the objective findings of the neurological examination supported by the electroencephalogram.

## CONCLUSIONS

In a group of 27 cases of bronchogenic carcinoma studied during life both by clinical neurological examination and electroencephalogram, and with complete autopsy, brain involvement occurred in 15. The neurological examination failed to predict involvement in only one of these cases, while the electroencephalogram missed 6 (4 of them in the cerebellum).

In 2 additional cases where the brain was uninvolved, the electroencephalogram suggested lateralized intracranial abnormality while the neurological examination was normal.

In 4 of the patients who presented clinical evidence of brain involvement, this was the only apparent indication that the disease had spread beyond the lung.

This study indicates the value of a careful neurological evaluation of prospective candidates for removal of a malignant lung tumor. It seems clear, moreover, that while the electroencephalogram is a useful aid in confirmation it should not be depended upon in itself as a screening technique.

## REFERENCES

1. Adler, cited by Ewing: Neoplastic Diseases, Philadelphia, 1940, W. B. Saunders Company.
2. Simpson, cited by Wilson, S. A. K.: Neurology, ed. 2, Baltimore, 1955, Williams & Wilkins Company.
3. King, A. B., and Ford, F. R.: A Clinical and Anatomical Study of Neurological Conditions Resulting From Metastases in the Central Nervous System Due to Carcinoma of the Lung, Bull. Johns Hopkins Hosp. 70: 124-156, 1942.
4. Halpert, B., Fields, W. S., and DeBakey, M. E.: Intracranial Metastasis From Carcinoma of the Lung, Surgery 35: 346-349, 1954.
5. Smith, J. R., Walter, C. W. P., and Laidlaw, R. W.: Electroencephalogram in Cases of Neoplasms of Posterior Fossa, Arch. Neurol. & Psychiat. 43: 472, 1940.
6. Rheinberger, M. B., and Davidoff, L. M.: Posterior Fossa Tumours and Electroencephalogram, J. Mt. Sinai Hosp. 9: 731, 1942.
7. Pupe, P. P., Pazzanese, O., and Pimenta, A. M.: The EEG in the Diagnosis of Tumours of the Brain, Tr. Am. Neurol. Assoc. 77: 252, 1952.
8. Daly, D., Whelan, J. L., Bickford, R. G., and MacCarty, C. S.: The EEG in Cases of Tumours of the Posterior Fossa and Third Ventricle, Electroencephalog. & Clin. Neurophysiol. 5: 203, 1953.
9. Cuneo, H. M., Rand, C. W., and Sjaardema, H.: Analysis of Electroencephalographic Findings in 40 Cases of Verified Brain Tumour, Bull. Los Angeles Neurol. Soc. 14: 1, 86, 149, 1949, and 15: 22, 1950.
10. Henson, R. A., Russell, D., and Wilkinson, M.: Carcinomatous Neuropathy and Myopathy, Brain 77: 82, 1954.

# The American Association for Thoracic Surgery

1957-1958

## Officers

President, Brian Blades, Washington, D. C.
Vice President, Michael E. DeBakey, Houston, Texas
Treasurer, Julian Johnson, Philadelphia, Pa.
Secretary, Hiram T. Langston, Chicago, Ill.
Editor, Emile Holman, San Francisco, Calif.

## Council

William E. Adams (1958)
Warriner Woodruff (1959)
Cameron Haight (1958)
H. Brodie Stephens (1960)
Frederick G. Kergin (1961)

## Membership Committee

Chairman, Duane Carr

Edward F. Beattie, Jr.
Edouard D. Gagnon
Frank L. Gerbode
Herbert C. Maier
Laurence M. Shefts
John W. Strieder

## Association Representatives on the Board of Thoracic Surgery

Julian Johnson
John C. Jones
Edward M. Kent
William M. Tuttle

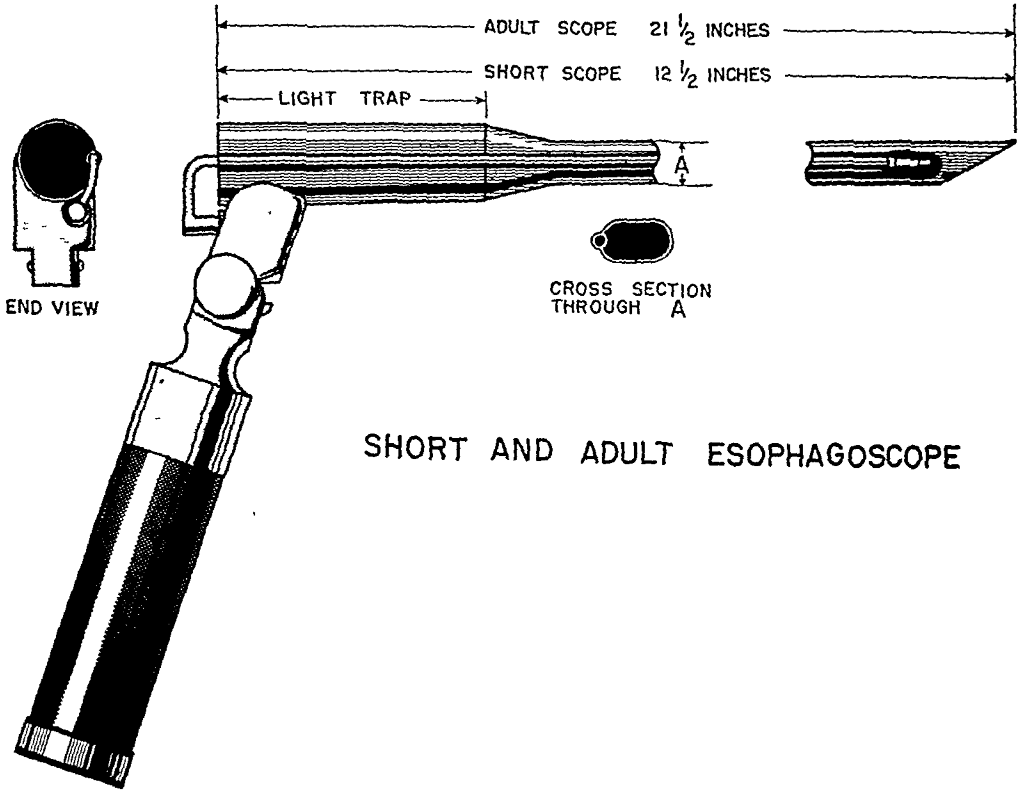


Fig. 1.—Diagrammatic drawing of ribbon esophagoscopes.

## CONCLUSION

A brief presentation of a new ribbon esophagoscope* has been made.

It has been found to be satisfactory for the purpose for which it was designed.

Certain advantages inherent in such a scope have been pointed out, such as, ease and safety of insertion, no free wires to entangle operator or his aide, light trap, and diagonal instrumentation.

## REFERENCE

1. Emerson, E. B., Jr.: Intraluminal Resection of Esophageal Strictures, A. M. A. Arch. Surg. 71: 863-866, 1955.

---

*The ribbon esophagoscopes are available to the profession at Welch Allyn, Inc., Skaneateles Falls, New York.

Boyd, David P. ........ 605 Commonwealth Ave., Boston, Mass.
Bradshaw, Howard H. ........ Bowman Gray School of Medicine, Winston Salem, N. C.
Brantigan, Otto C. ........ 101 W. Madison St., Baltimore, Md.
Brewer, Lyman A., III ........ 2010 Wilshire Blvd., Los Angeles 57, Calif.
Brindley, George V., Jr. ........ Scott and White Clinic, Temple, Texas
Brown, A. Lincoln ........ 490 Post Street, San Francisco, Calif.
Brown, Robert K. ........ 1624 Gilpin St., Denver 6, Colo.
Brownrigg, Garrett M. ........ 47 Queens Road, St. Johns, Newfoundland
Buckingham, William W. ........ 314 Professional Bldg., Kansas City, Mo.
Bugden, Walter F. ........ Medical Arts Bldg., Syracuse 10, N. Y.
Burford, Thomas H. ........ Barnes Hospital, St. Louis 10, Mo.
Burnett, W. Emory ........ Broad and Ontario Sts., Philadelphia, Pa.
Byron, Francis X. ........ 120 S. Lasky Dr., Suite 203, Beverly Hills, Calif.

Carlson, Robert I. ........ Veterans Adm. Hospital, Nashville, Tenn.
Carr, Duane ........ 899 Madison Ave., Memphis, Tenn.
Carter, Max G. ........ 6[illegible] George St., New Haven, Conn.
Chamberlain, John Maxwell ........ 23 East 79th St., New York 21, N. Y.
Chambers, John S., Jr. ........ 233 "A" St., Room 1205, San Diego 1, Calif.
Chesney, John G. ........ 2300 Biscayne Blvd., Miami 37, Fla.
Clagett, O. T. ........ Mayo Clinic, Rochester, Minn.
Clowes, George H. A., Jr. ........ 3395 Scranton Road, Cleveland 9, Ohio
Cohn, Roy B. ........ Stanford University Hospital, San Francisco, Calif.
Coleman, Frank P. ........ 810 W. Franklin St., Richmond, Va.
Condon, William B. ........ 110 Republic Bldg., Denver, Colo.
Conklin, William S. ........ 1016 Standard Ins. Bldg., Portland 5, Ore.
Cooley, Denton A. ........ Baylor University College of Medicine, Houston 25, Texas
Cooper, David A. ........ 1520 Spruce St., Philadelphia, Pa.
Cotton, Bert H. ........ 1321 N. Vermont Ave., Los Angeles 27, Calif.
Cournand, Andre ........ Bellevue Hospital, 27th St. and 1st Ave., New York 16, N. Y.
Crandell, Walter B. ........ Veterans Adm. Hospital, White River Junction, Vt.
Creech, Oscar, Jr. ........ Tulane University School of Medicine, New Orleans 12, La.
Crimm, Paul D. ........ Boehne Hospital, Evansville 12, Ind.
Curreri, Anthony R. ........ 1300 University Ave., Madison, Wis.

Dailey, James E. ........ 4109 Montrose Blvd., Houston 6, Texas
Daniel, Rollin A. ........ 410 Medical Arts Bldg., Nashville, Tenn.
Daniels, Albert C. ........ 490 Post St., San Francisco 2, Calif.
Daughtry, DeWitt C. ........ 4201 Lake Road, Bay Point, Miami, Fla.
Davis, Edgar W. ........ 1150 Connecticut Ave., Washington, D. C.
Day, J. Claude ........ 307 David Whitney Bldg., Detroit 26, Mich.
DeBakey, Michael E. ........ Baylor University, Dept. of Surgery, Houston, Texas
DeCamp, Paul T. ........ 3503 Prytania Street, New Orleans 15, La.
Delarue, Norman C. ........ 25 Donlea Drive, Toronto 7, Ont.
Dennis, Clarence E. ........ 989 Edgewood Ave., Pelham Manor, N. Y.
Deshaies, Georges ........ 37 Bellingham Road, Montreal, Que.
Deterling, Ralph A. ........ 180 Ft. Washington Ave., New York, N. Y.
Dodrill, Forest D. ........ 621 David Whitney Bldg., Detroit, Mich.
Domm, Sheldon E. ........ 1918 W. Clinch Ave., Knoxville 16, Tenn.
Dorner, Ralph A. ........ 710 Equitable Bldg., Des Moines 9, Iowa
Dorsey, John M. ........ 636 Church St., Evanston, Ill.
Douglass, Richmond ........ Veterans Adm. Hospital, Castle Point, N. Y.
Drake, Emerson H. ........ 18 Bramhall St., Portland 3, Maine
Drash, Everett C. ........ University of Virginia Hospital, Charlottesville, Va.
Dugan, David J. ........ 459—30th St., Oakland 9, Calif.

# LIST OF MEMBERS* OF THE AMERICAN ASSOCIATION FOR THORACIC SURGERY, 1957-58

## Honorary Members

ALLISON, PHILIP ---- Radcliffe Infirmary, Oxford, England
BROCK, SIR RUSSELL C. ---- Guy's Hospital, London, England
CRAFOORD, CLARENCE ---- Sabbatsberg Sjukhus, Stockholm, Sweden
DAVIES, H. MORRISTON ---- Broadgreen Hospital, Edgelane Drive, Liverpool 14, England
DENK, WOLFGANG ---- Surgical University Clinic, Vienna, Austria
JACKSON, CHEVALIER ---- Old Sunrise Mills, Schwenkville, Pa.
KELLER, WILLIAM L., COL., MC, USA, (RET.) ---- 2930 Foxhall Road, N.W., Washington, D. C.
LERCHE, WILLIAM ---- Larkhills, Cable, Wis.
MATAS, RUDOLPH ---- 2255 St. Charles Ave., New Orleans, La.
SEMB, CARL ---- Ullevaal Hospital, Oslo, Norway
SHENSTONE, NORMAN S. ---- 904 Medical Arts Bldg., Toronto 5, Ont.
THOMAS, SIR CLEMENT PRICE ---- 69 Harley St., London, W. 1, England

## Active Members

ABBOTT, OSLER ---- Emory University Hospital, Emory University, Ga.
ADA, ALEXANDER E. W. ---- 139 East 94th St., New York, N. Y.
ADAMS, HERBERT D. ---- Lahey Clinic, 605 Commonwealth Ave., Boston, Mass.
ADAMS, RALPH ---- Huggins Hospital, Wolfeboro, N. H.
ADAMS, WILLIAM E. ---- University of Chicago, 950 East 59th St., Chicago 37, Ill.
ALLBRITTEN, FRANK F., JR. ---- University of Kansas Medical Center, Kansas City 12, Kan.
ALLEY, RALPH D. ---- Albany Hospital, Albany, N. Y.
ASHBURN, FRANK S. ---- 1835 Eye St., N.W., Washington, D. C.
AUERBACH, OSCAR ---- Veterans Adm. Hospital, East Orange, N. J.
AUFSES, ARTHUR H. ---- 1158 Fifth Ave., New York 29, N. Y.

BADGER, THEODORE ---- 264 Beacon St., Boston 16, Mass.
BAHNSON, HENRY T. ---- 201 Cedarcroft Road, Baltimore 12, Md.
BAILEY, CHARLES P. ---- 219 N. Broad St., Philadelphia 7, Pa.
BALLON, HARRY C. ---- 1414 Drummond St., Montreal, Que.
BARKLEY, HOWARD T. ---- 4109 Montrose Blvd., Houston 6, Texas
BARONOFSKY, IVAN D., CDR. (MC), USNR ---- U.S. Naval Hospital, San Diego 34, Calif.
BEATTIE, EDWARD J., JR. ---- 1753 W. Congress St., Chicago 12, Ill.
BEECHER, HENRY K. ---- Massachusetts General Hospital, Boston 14, Mass.
BENEDICT, EDWARD B. ---- Massachusetts General Hospital, Boston 14, Mass.
BENSON, CLIFFORD D. ---- 1515 David Whitney Bldg., Detroit, Mich.
BETTS, REEVE H. ---- Christian Medical College, Vellore, So. India
BISGARD, J. DEWEY ---- 1420 Medical Arts Bldg., Omaha, Neb.
BLACK, HARRISON ---- 67 Bay State Road, Boston 15, Mass.
BLADES, BRIAN ---- 901 Twenty-third St., N.W., Washington, D. C.
BORTONE, FRANK ---- 2765 Hudson Blvd., Jersey City 6, N. J.
BOSHER, LOUIS H. ---- 1200 E. Broad St., Richmond, Va.

---

*If any corrections should be made in the spelling of names or in the addresses of members, please notify the Administrative Assistant of the Association, Miss Ada Hanvey, 600 South Kingshighway, St. Louis 10, Mo.

Johnson, Elgie K ----- 230 Hilton St, Hempstead, N Y
Johnson, Hollis E ----- 2122 West End Ave, Nashville, Tenn
Johnson, Julian ----- 3400 Spruce St, Philadelphia, Pa
Johnston, James H, Jr ----- 710 N State St, Jackson, Miss
Jones, John C ----- 1136 West 6th St, Los Angeles, Calif
Joynt, G H C ----- 399 Bathurst St, Toronto, Ont.

Kay, Earle B ----- 10465 Carnegie Ave, Cleveland, Ohio
Keeley, John L ----- 30 North Michigan Ave, Chicago 2, Ill
Kent, Edward M ----- 3500 Fifth Ave, Pittsburgh 13, Pa
Kergin, Frederick G ----- Medical Arts Bldg, Toronto 5, Ont
Kessler, Charles R ----- 1027 Woodward Bldg, Birmingham 3, Ala
King, Richard ----- 814 Doctors Bldg, Atlanta, Ga
Kinsella, Thomas J ----- 1251 Medical Arts Bldg, Minneapolis, Minn
Kipp, Harold A ----- Mercy Hospital, Pittsburgh 15, Pa
Kirby, Charles K ----- 3400 Spruce Street, Philadelphia, Pa
Kirklin, John W ----- Mayo Clinic, Rochester, Minn
Klassen, Karl P ----- Ohio State University, Columbus, Ohio
Klepser, Roy G ----- 1835 Eye St, N W, Washington, D C
Klopstock, Robert ----- Veterans [illegible] Hospital, Brooklyn 9, N Y

Laird, Robert ----- 399 Bathurst St, Toronto, Ont
Lam, Conrad R ----- Henry Ford Hospital, Detroit, Mich
Lambert, Adrian ----- [illegible] Park Ave, New York, N Y
Lampson, R Starr ----- 8 Jefferson St, Hartford, Conn
Langston, Hiram T ----- 1919 West Taylor St, Chicago 12, Ill
Lauley, James R ----- 5710 16th St, N W, Washington, D C
Leeds, Sanford E ----- 2211 Post St, San Francisco 15, Calif
Lees, William M ----- 7000 Kenton St, Lincolnwood 30, Ill
Lester, Charles W ----- 70 East 80th St, New York, N Y
Leven, N Logan ----- 1464 Lowry Medical Arts Bldg, St Paul, Minn
Lewis, F John ----- Northwestern Univ Med School, Chicago 11, Ill
Lillehei, Clarence W ----- University Hospitals, Minneapolis 14, Minn
Lindskog, Gustaf E ----- 50 Marvel Road, New Haven, Conn
Longmire, William P, Jr ----- UCLA School of Medicine, Los Angeles 24, Calif
Lynch, Joseph P ----- 1180 Beacon St, Brookline, Mass

Mackler, Saul A ----- 104 S Michigan Ave, Chicago 3, Ill
MacManus, Joseph ----- 491 Delaware, Buffalo, N Y
Maier, Herbert C ----- 3 East 71st St, New York, N Y
Major, Robert C ----- University Hospital, Augusta, Ga
Mannix, Edgar P, Jr ----- 12 Forest Turn, Manhasset, Long Island, N Y
Maurer, Elmer P R ----- 827 Union Central Bldg, Cincinnati, Ohio
Mautz, F R ----- 10515 Carnegie Ave, Cleveland 6, Ohio
Mayer, John H, Jr ----- 503 Plaza Parkway Bldg, Kansas City, Mo
McDonald, John R ----- Mayo Clinic, Rochester, Minn
McIntosh, Clarence A ----- 1390 Sherbrooke St, W, Montreal, Que
Meltzer, Herbert ----- 505 Medical Arts Bldg, Edmonton, Alberta, Canada
Merendino, K Alvin ----- University of Washington, Seattle, Wash
Merker, Carl G ----- 8 Church St, Saranac Lake, N Y
Mills, Waldo O ----- 1445 Medical and Dental Bldg, Seattle 1, Wash
Minor, George F ----- Univ of Virginia Hospital, Charlottesville, Va
Miscall, Laurence ----- 11 East 68th St, New York, N Y

Effler, Donald B.……Euclid and East 93rd Sts., Cleveland, Ohio
Ehrenhaft, Johann L.……University of Iowa, Iowa City, Iowa
Ellis, F. Henry, Jr.……Mayo Clinic, Rochester, Minn.
Ellison, Robert G.……Medical College of Georgia, Augusta, Ga.
Evans, Byron H.……2940 Fresno St., Fresno, Calif.

Falor, William H.……623 Second National Bldg., Akron 8, Ohio
Fell, Egbert H.……122 So. Michigan Ave., Chicago 3, Ill.
Fischer, Walter W.……170 East 78th St., New York, N. Y.
Ford, William B.……3500 Fifth Ave., Pittsburgh 13, Pa.
Forsee, James H., Col., MC, USA……5207 Falmouth Road, Washington 16, D.C.
Frank, Howard A.……330 Brookline Ave., Boston 15, Mass.
Freelander, Samuel O.……2460 Fairmount Blvd., Cleveland Heights 6, Ohio

Gaensler, Edward A.……229 Dudley Rd., Newton Centre 59, Mass.
Gagnon, Edouard D.……902 Est., Rue Sherbrooke, Montreal, Que.
Gale, Joseph W.……Wisconsin General Hospital, Madison 6, Wis.
Garlock, John H.……3 E. 73rd St., New York, N. Y.
Geary, Paul……909 Park Ave., Plainfield, N. J.
Gebauer, Paul……Leahi Hospital, 649 Pokole St., Honolulu, T.H.
Gerbode, Frank L.……Stanford University Hospital, San Francisco, Calif.
Gibbon, John H., Jr.……1025 Walnut St., Philadelphia, Pa.
Glover, Robert P.……269 South 19th St., Philadelphia, Pa.
Gordon, Joseph……106 Girard Blvd., S.E., Albuquerque, N. M.
Grace, Archibald J.……530 Wellington St., London, Ont.
Grimes, Orville F.……University of California Hosp., San Francisco 22, Calif.
Gross, Robert E.……300 Longwood Ave., Brookline, Mass.
Grow, John B.……3705 E. Colfax, Denver, Colo.

Haight, Cameron……University Hospital, Ann Arbor, Mich.
Hanlon, Rollins……1401 S. Grand Blvd., St. Louis 4, Mo.
Harken, Dwight E.……67 Bay State Road, Boston 15, Mass.
Harper, Frederick R.……1104 Republic Bldg., Denver, Colo.
Harrison, Albert W.……Medical Branch, Univ. of Texas, Galveston, Texas
Harrison, Elliott……1862 West Broadway, Vancouver, B. C.
Harrison, Harlon W.……U.S. Naval Hospital, San Diego, Calif.
Harter, John S.……212 Brown Bldg., Louisville 2, Ky.
Head, Jerome R.……55 E. Washington St., Chicago, Ill.
Helmsworth, James A.……Cincinnati General Hospital, Cincinnati 29, Ohio
Higginson, John F.……2455 N. W. Marshall St., Portland, Ore.
Himmelstein, Aaron……70 East 96th St., New York 28, N. Y.
Hochberg, Lew A.……135 Eastern Parkway, Brooklyn 17, N. Y.
Holinger, Paul H.……700 N. Michigan Ave., Chicago, Ill.
Holman, Cranston W.……862 Fifth Ave., New York 21, N. Y.
Hopkins, William A.……710 Peachtree Street, Atlanta, Ga.
Hudson, Theodore R.……55 E. Washington St., Chicago, Ill.
Hughes, Felix A., Jr.……Kennedy Hospital, Memphis, Tenn.
Humphreys, George H.……180 Ft. Washington Ave., New York, N. Y.
Hurley, Gerard A. P.……1538 Sherbrooke St., W., Montreal, Que.
Hurwitt, Elliott S.……Montefiore Hospital, New York 67, N. Y.
Hurwitz, Alfred……Maimonides Hosp., 4802 Tenth Ave., Brooklyn, N. Y.

Jackson, Chevalier L.……3401 N. Broad St., Philadelphia 40, Pa.
Janes, Ernest C.……250 Main St., Hamilton, Ont.
Jarvis, Fred J.……819 Boylston Ave., Seattle 4, Wash.

Strieder, John W ---------- 1180 Beacon St, Brookline, Mass
Strode, Joseph E ---------- 1021 Kapiolani, Honolulu 14, T H
Swan, Henry, II ---------- 4200 East 9th Ave, Denver 20, Colo
Sweet, Richard H ---------- 87 Chestnut Street, Boston 8, Mass

Thompson, Samuel A ---------- 850 Park Ave, New York, N Y
Touroff, Arthur S W ---------- 994 Fifth Ave, New York 28, N Y
Tuttle, William M ---------- 1307 David Whitney Bldg, Detroit 26, Mich

Van Hazel, Willard ---------- 24 S Michigan Blvd, Chicago, Ill
Varco, Richard L ---------- University Hospital, Minneapolis, Minn
Vineberg, Arthur M ---------- 1390 Sherbrooke St, W, Montreal, Que
Vorwald, Arthur J ---------- College of Medicine Wayne University, Detroit 7, Mich

Waddell, William R ---------- 69 Woodland Road Chestnut Hill, Mass
Walker, James H ---------- 1123 Virginia St, E Charleston, W Va
Ware, Paul F ---------- 16 Norwich St Worchester, Mass
Waterman, David H ---------- 1918 West Clinch Ave Knoxville 16, Tenn
Watson, William L ---------- 1[illegible] Park Ave, New York, N Y
Webb, Watts R ---------- University Hospital, Jackson, Miss
Weisel, Wilson ---------- 324 E Wisconsin Ave Milwaukee 2, Wis
White, Marion L, Jr ---------- Huntington Bank Bldg, Huntington, W Va
Willauer, George ---------- 1930 Chestnut St, Philadelphia, Pa
Williams, Mark H ---------- 6 Front St Binghamton, N Y
Wilson, Julius L ---------- Henry Phipps Institute, Philadelphia, Pa
Wilson, Norman J ---------- 13 Francis St Boston 15, Mass
Wiper, Thomas B ---------- 336 Mason St, San Francisco, Calif
Woodruff, Wardner ---------- 8 Church St, Saranac Lake, N Y
Woods, Francis M ---------- 13[illegible] Francis St, Boston 15, Mass
Wright, George W ---------- 11311 Shaker Blvd Cleveland 4, Ohio
Wylie, Robert H ---------- 903 Park Ave, New York, N Y

### Associate Members

Ackman, F Douglas ---------- 1374 Sherbrooke St, W, Montreal, Que
Adelman, Arthur ---------- 701 East 63rd St, Kansas City, Mo
Adler, Richard H ---------- 100 High St, Buffalo 3, N Y
Aitchison, David B ---------- Mountain Sanatorium, Hamilton, Ont
Andrews, Neil C ---------- Ohio Tuberculosis Hospital, Columbus 10, Ohio

Baffes, Thomas G ---------- Children s Memorial Hospital, Chicago 14, Ill
Battersby, James S ---------- 1040 W Michigan St, Indianapolis, Ind
Bell, John W ---------- Veterans Adm Hosp, 4435 Beacon Ave, Seattle 8, Wash
Benoit, Hector W, Jr ---------- 503 Plaza Parkway Bldg, Kansas City, Mo
Berg, Ralph, Jr ---------- 231 Medical Center Bldg, Spokane 4, Wash
Bergmann, Martin ---------- 4500 Olive St, St Louis 8, Mo
Bernatz, Philip E ---------- Mayo Clinic, Rochester, Minn
Bloomberg, Allan E ---------- 1095 Park Ave, New York 28, N Y
Bousquet, Ernest O ---------- 5689 Boulevard Rosemont, Montreal, Que
Bruneau, Jacques ---------- 847 Cherrier, Montreal 24, Que
Bryant, Joseph R ---------- 321 West Broadway, Louisville 2, Ky
Burbank, Benjamin ---------- 244 Henry St, Brooklyn 2, N Y

Cahan, William G ---------- 444 E 68th St, New York 21, N Y
Chandler, John H ---------- 616 W Forrest, The Jackson Clinic, Jackson, Tenn
Chodoff, Richard J. ---------- 255 South 17th St, Philadelphia, Pa.

| | |
|---|---|
| Mudd, James L. | 634 N. Grand Blvd., St. Louis 3, Mo. |
| Muller, Wm. H., Jr. | Univ. of Virginia Hospital, Charlottesville, Va. |
| Mulvihill, Daniel A. | 15 East 77th St., New York, N. Y. |

| | |
|---|---|
| Oatway, William H., Jr. | La Vina Sanatorium, Altadena, Calif. |
| Ochsner, Alton | Tulane Univ. School of Medicine, New Orleans, La. |
| Olsen, Arthur M. | 102 2nd Ave., S.W., Rochester, Minn. |
| O'Neill, Thomas J. | 269 South 19th St., Philadelphia 3, Pa. |
| O'Rourke, Paul V. | 307 David Whitney Bldg., Detroit, Mich. |
| Overholt, Richard H. | 135 Francis St., Boston 15, Mass. |

| | |
|---|---|
| Paine, John R. | The Buffalo General Hosp., 100 High St., Buffalo, N. Y. |
| Papper, Emmanuel M. | 622 West 168th St., New York 32, N. Y. |
| Parker, Edward F. | 86 Hasell St., Charleston, S. C. |
| Paulson, Donald L. | 3810 Swiss Ave., Dallas, Texas |
| Peters, Richard M. | University of North Carolina, Chapel Hill, N. C. |
| Pool, John L. | 755 Park Ave., New York 21, N. Y. |
| Poppe, J. Karl | 1130 S. W. Morrison St., Portland, Ore. |
| Potts, Willis J. | 707 Fullerton Ave., Chicago 14, Ill. |
| Proctor, Oscar S. | Box 1053, Topeka, Kan. |

| | |
|---|---|
| Raine, Forrester | 425 E. Wisconsin Ave., Milwaukee, Wis. |
| Ramsey, Beatty H. | 2210 Santa Monica Blvd., Santa Monica, Calif. |
| Rasmussen, Richard A. | Blodgett Medical Bldg., Grand Rapids, Mich. |
| Ravitch, Mark M. | Johns Hopkins Hospital, Baltimore, Md. |
| Read, Charles T. | 550 West Thomas Road, Phoenix, Ariz. |
| Richards, Victor | Stanford-Lane Hospital, San Francisco 15, Calif. |
| Riggins, H. McLeod | 140 East 54th St., New York, N. Y. |
| Ripstein, Charles B. | 54 Rose Ave., Great Neck, L. I., N. Y. |
| Robertson, Ross | 925 West Georgia St., Vancouver 1, B. C. |
| Rogers, W. L. | 490 Post St., San Francisco, Calif. |
| Rosemond, George P. | 3401 Broad St., N., Philadelphia, Pa. |
| Rumel, William R. | 807 Medical Arts Bldg., Salt Lake City, Utah |

| | |
|---|---|
| Samson, Paul C. | 3959 Happy Valley Road, Lafayette, Calif. |
| Sanger, Paul W. | Doctors Bldg., Kings Drive, Charlotte, N. C. |
| Sarot, Irving A. | 111 East 69th St., New York 21, N. Y. |
| Scannell, J. Gordon | Massachusetts General Hospital, Boston, Mass. |
| Schaffner, Vernon D. | Kentville, Nova Scotia |
| Schmidt, Herbert Wm. | Mayo Clinic, Rochester, Minn. |
| Scott, Henry W., Jr. | Vanderbilt University Hospital, Nashville, Tenn. |
| Sealy, Will C. | Duke University Hospital, Durham, N. C. |
| Seley, Gabriel | 540 Park Ave., New York 21, N. Y. |
| Shaw, Robert R. | 3810 Swiss Ave., Dallas, Texas |
| Shefts, Laurence M. | 503 Moore Bldg., San Antonio, Texas |
| Shumacker, Harris, B., Jr. | Indiana Univ. Medical Center, Indianapolis, Ind. |
| Skinner, Edward F. | 899 Madison Ave., Memphis, Tenn. |
| Skinner, George F. | 36 Coburg St., St. Johns, N. B. |
| Sloan, Herbert | University Hospital, Ann Arbor, Mich. |
| Sommer, George N. J., Jr. | 120 W. State St., Trenton, N. J. |
| Soutter, Lamar | 80 East Concord St., Boston, Mass. |
| Steele, J. D. | Veterans Adm. Hospital, San Fernando, Calif. |
| Stephens, H. Brodie | 384 Post St., San Francisco, Calif. |
| Storey, Clifford F. | 1309 Medical-Dental Bldg., San Diego 1, Calif. |
| Stranahan, Allen | Albany Hospital, Albany, N. Y. |

JAHNKE, EDWARD J, JR ---- Walter Reed Army Hospital, Washington 12, D C
JAMPLIS, ROBERT W ---- 300 Homer Ave, Palo Alto, Calif
JENSEN, NATHAN K ---- 1629 Medical Arts Bldg, Minneapolis 2, Minn
JENSIK, ROBERT J ---- 224 S Michigan Ave, Chicago 4, Ill
JOHNS, THOMAS N P ---- 6305 Lowma Road, Richmond, Va
JOHNSON, CLIVE R ---- 1216 Pennsylvania Ave, Fort Worth 4, Texas
JOHNSTON, FRANK R ---- Bowman Gray School of Med, Winston Salem, N C
JUDD, ARCHIBALD R ---- 304 N Fourth St, Hamburg, Pa
JULIAN, ORMAND C ---- 25 E Washington St, Chicago, Ill

KAUNITZ, VICTOR H ---- 685 Delaware Ave, Buffalo 9, N Y
KELLEY, WINFIELD O ---- Uncas on Thames, Norwich, Conn
KEMLER, R LEONARD ---- 576 Farmington Ave, Hartford 5, Conn
KENNEY, LEO J ---- 610 Medical Arts Bldg, Grand Rapids 2, Mich
KIRSCHNER, PAUL A ---- 2 East 92nd St, New York 28, N Y
KITTLE, C FREDERICK ---- University of Kansas Medical Center, Kansas City, Kan
KRAEFT, NELSON H ---- 1501 Magnolia Drive, Tallahassee, Fla
KUNDERMAN, PHILIP J ---- 165 Livingston Ave, New Brunswick N J
KUNSTLER, WALTER L ---- 1538 Sherbrooke St, W, Montreal 25, Que

LEETCH, HENRY W ---- 108 Main St, Saranac Lake, N Y
LEIBOVITZ, MARTIN ---- 812 Medical Arts Bldg, Tulsa, Okla
LEWIS, J EUGENE, JR ---- 1325 South Grand Blvd, St Louis 4, Mo
LEWIS, RUBIN M ---- 380 Ellsworth, Berkeley, Calif
LONGACRE, JACOB J ---- 150 Carew Tower, Cincinnati, Ohio
LUCIDO, JOSEPH L ---- 634 North Grand, St Louis 3, Mo
LYON, CLAYTON ---- 384 Post St, San Francisco, Calif

MACDONALD, NEIL ---- Medical Arts Bldg, Windsor, Ont
MACPHERSON, LACHLAN ---- St John Tuberculosis Hospital, East St John, N B
MADER, VICTOR O ---- 149 S Park St, Halifax, Nova Scotia
MADOFF, IRVING M ---- 1180 Beacon St, Brookline 46, Mass
MANGIARDI, JOSEPH L ---- 520 Franklin Ave, Garden City, N Y
MASON, JAMES M, III ---- 1023 South 20th St Birmingham, Ala
MCBURNEY, ROBERT P ---- 899 Madison Ave, Memphis 3, Tenn
MELICK, DERMONT W ---- 1005 Professional Bldg, Phoenix, Ariz
MENDELSOHN, HARVEY J ---- 2065 Adelbert Road, Cleveland, Ohio
MENDELSSOHN, EDWIN ---- 1301 Tabor Road, Philadelphia, Pa
MEYER, BERTRAND W ---- 1136 West Sixth St, Los Angeles, Calif
MICHELSON, ELLIOTT ---- 1801 Eutaw Place, Baltimore, Md
MILLER, CARROLL C ---- 304 Humphrey St, Swampscott, Mass
MORROW, ANDREW G ---- National Heart Institute, Bethesda 14, Md
MOULDER, PETER V ---- 950 E 59th St, Chicago 37, Ill
MOUSEL, LLOYD H ---- Dept of Anesthesiology, The Swedish Hospital, Seattle, Wash

NEMIR, PAUL, JR ---- University of Pennsylvania, Philadelphia 4, Pa
NEPTUNE, WILFORD B ---- 135 Francis St, Boston 15, Mass
NEWMAN, MELVIN M ---- State Univ of N Y, 450 Clarkson Ave, Brooklyn 3, N Y
NEWMAN, ROBERT W ---- Medical Arts Bldg, Knoxville, Tenn

O'NEILL, JAMES F ---- 140 Roslyn Ave, Glenside, Pa

PAUL, JOHN S ---- Baker VA Center, Martinsburg, W Va
PHILLIPS, FRANCIS J ---- Seward Sanatorium, Bartlett, Alaska
PINKHAM, ROLAND D ---- Suite 1445, Medico Dental Bldg, Seattle, Wash

Chunn, Charles F.--------442 W. Lafayette St., Tampa 6, Fla.
Cincotti, John J.--------Veterans Adm. Hospital, Brooklyn 9, N. Y.
Clatworthy, H. William, Jr.--------The Children's Hosp., 561 South 17th, Columbus, Ohio
Connar, Richard G.--------706 Franklin St., Tampa 2, Fla.
Cooke, Francis N.--------25 S. E. Second Ave., Miami, Fla.
Cox, William V.--------133 Court St., Auburn, Maine
Cracovaner, Arthur J.--------103 East 78th St., New York 21, N. Y.
Crastnopol, Philip--------1221 East 21st St., Brooklyn, N. Y.
Crecca, Anthony D.--------376 Roseville Ave., Newark 7, N. J.
Cross, Frederick S.--------10465 Carnegie Ave., Cleveland, Ohio
Cutler, Preston R.--------807 Medical Arts Bldg., Salt Lake City 1, Utah

Dafoe, Colin S.--------508 Medical Arts Bldg., Edmonton, Alberta
Dammann, John F.--------632 Preston Place, Charlottesville, Va.
Dasch, Frederick W.--------416 West Market St., Pottsville, Pa.
Deaton, W. Ralph, Jr.--------1027 Professional Village, Greensboro, N. C.
DeBord, Robert A.--------105 Northland Ave., Peoria, Ill.
Decker, Alfred M., Jr.--------Sunmount, N. Y.
DeMatteis, Albert--------1216—13th Ave., Altoona, Pa.
Diveley, Walter L.--------410 Medical Arts Bldg., Nashville 2, Tenn.
Dodds, G. Alfred--------807 Broadway, Fargo, N. D.

Eglee, Edward P.--------105 East 53rd St., New York, N. Y.
Emerson, George L.--------26 Strathallan Park, Rochester 7, N. Y.

Finnerty, James--------Brookhaven Medical Arts Bldg., Patchogue, N. Y.
Fishback, Frederick C.--------1835 Eye St., N.W., Washington, D. C.
Fox, Robert--------2136 Robincrest Lane, Glenview, Ill.
French, Sanford W., III--------Letterman Army Hospital, San Francisco, Calif.
Friedlander, Ralph--------The Bronx Hospital, Bronx 56, N. Y.
Friesen, Stanley R.--------39th and Rainbow, Kansas City 3, Kan.
Fuller, Josiah--------205 W. 2nd St., Duluth 2, Minn.

Gerbasi, Francis S.--------3637 Vermont, Long Beach 14, Calif.
Glenn, Frank--------525 East 68th St., New York 21, N. Y.
Glenn, William W. L.--------333 Cedar St., New Haven, Conn.
Goldman, Alfred--------416 N. Bedford Drive, Beverly Hills, Calif.
Grace, Edwin J.--------121 Fort Green Place, Brooklyn, N. Y.
Gravel, Joffre-Andre--------11 Place George Vth, Quebec City, Que.
Greer, Allen E.--------1200 North Walker, Oklahoma City, Okla.
Groves, Laurence K.--------Cleveland Clinic, Cleveland 6, Ohio
Gwathmey, Owen--------901 23rd St., N.W., Washington 7, D. C.

Hampton, Foster, Jr.--------Suite 101, Interstate Bldg., Chattanooga, Tenn.
Hanner, Joseph M.--------U. S. Naval Hospital, San Diego 34, Calif.
Hardy, James D.--------University of Mississippi Medical Center, Jackson, Miss.
Hausmann, Paul F.--------2212 West State St., Milwaukee 3, Wis.
Herben, George F.--------House of Rest at Sprain Ridge, Yonkers, N. Y.
Heroy, William W.--------22 Oakwood Road, Huntington, N. Y.
Herrera-Llerandi, Rodolfo E.--------Centro Medico, Guatemala City, Guatemala, C. A.
Hertzler, Jack H.--------4377 West Maple Road, Birmingham, Mich.
Hun, Henry--------149 Washington Ave., Albany, N. Y.

Ingram, Ivan N.--------350 Post St., San Francisco 8, Calif.
Iovine, Vincent M.--------1150 Connecticut Ave., N.W., Washington, D. C.

Ballon, David — 1538 Sherbrooke St, W, Montreal 25, Que
Barnwell, John B — Room 866, Veterans Adm, Washington 25, D C
Bazin, A T — 1414 Drummond St, Montreal, Que
Beck, Claude S — 2065 Adelbert Road, Cleveland, Ohio
Berry, Frank B — 1301 Massachusetts Ave, Washington 16, D C
Bettman, Ralph B — 104 S Michigan Ave, Chicago, Ill
Bird, Clarence E — 64 Alfred Stone Rd, Providence 6, R I
Blalock, Alfred — Johns Hopkins Hospital, Baltimore, Md
Bloch, Robert G — Montefiore Hospital, New York 67, N Y
Butler, Ethan Flagg — [illegible]56 West Water Street, Elmira, N Y
Byers, H Roddick — Gananoque, Ont

Carlson, Herbert A — [illegible] East 14th St, Long Beach, Calif
Carter, B Noland — Cincinnati General Hospital, Cincinnati, Ohio
Churchill, Edward D — Massachusetts General Hospital, Boston, Mass
Clerf, Louis H — 5575 Eighth Ave, North St Petersburg 2, Fla
Cole, Dean B — Professional Bldg, Richmond, Va
Curtis, George M — Ohio State Univ College of Medicine, Columbus, Ohio

Davidson, Louis R — 30 East [illegible]0th Street, New York 22, N Y
Decker, Harry R — 730 The Park Bldg, [illegible]—5th Ave, Pittsburgh 22, Pa
Dieffenbach, Richard H — 570 Mt Prospect Ave, Newark 4, N J
Dooley, Frank S — 2010 Wilshire Blvd, Los Angeles 57, Calif
Dovell, Chauncey, Col, MC, (Ret) — 62 South Boxwood St, Hampton, Va

Elkin, Daniel C — Elkin Place, Lancaster, Ky
Eloesser, Leo — 490 Post St, San Francisco, Calif

Faulkner, William B, Jr — 1802 Fillmore St, San Francisco, Calif
Ferguson, R G — Balfour Apts, Regina, Sask
Flick, John B — 225 South Fifteenth St, Philadelphia 2, Pa

Harrington, Stuart W — Mayo Clinic, Rochester, Minn
Hart, Deryl — Duke University, Durham, N C
Hayes, John N — 21 Church St, Saranac Lake, N Y
Heinbecker, Peter — Washington University Medical School, St Louis, Mo
Holman, Emile — 722 Funston Ave, San Francisco, Calif
Hudson, William A — 602 David Whitney Bldg, Detroit, Mich

Janes, Robert M — Medical Arts Bldg, Toronto, Ont
Johns, Frank S — Johnston Willis Hospital, Richmond, Va

Kernan, John D — 103 East 78th St, New York, N Y
King, Donald S — Hitchcock Clinic, Hanover, N H
Knoepp, Louis F — Veterans Adm Hospital, Alexandria, La

Leahy, Leon J — 105 Medical Arts Bldg, Buffalo, N Y
Lewald, Leon T — 1200 Fifth Avenue, New York, N Y
Lockwood, A L — 300 Bloor St, E, Toronto, Ont

Meade, Richard H — Blodgett Medical Bldg, Grand Rapids, Mich
Meyer, Herbert Willy — Box 507, Rancho Santa Fe, Calif
Miller, Robert T, Jr — Mountain Lake, Lake Wales, Fla
Moersch, Herman — 725 Tenth Ave, Rochester, Minn
Moore, Julian A — 404 Flatiron Bldg, Asheville, N C
Moore, Richmond L — 180 Ft W [illegible] New York, N Y

Polk, John W.----------604 Medical Arts Bldg., Springfield 4, Mo.
Pollock, William C., Col., MC, USA----------1336 Cherry St., Denver 8, Colo.
Potter, Benjamin P.----------821 Bergen Ave., Jersey City, N. J.
Pratt, Lawrence A.----------3919 *John R Street*, Detroit, Mich.

Quinlan, John J.----------Nova Scotia Sanatorium, Kentville, Nova Scotia

Ransdell, Herbert T., Jr.----------Louisville General Hospital, Louisville 2, Ky.
Robbins, S. Gwin----------899 Madison Ave., Memphis, Tenn.
Robinson, Joseph L.----------1136 West Sixth Street, Los Angeles 17, Calif.
Roe, Benson B.----------384 Post St., San Francisco 8, Calif.
Ross, Raleigh R.----------2 Medical Arts Square, Austin 5, Texas
Ryan, Bernard J.----------375 East Main St., Bay Shore, N. Y.
Ryan, Thomas C.----------90 Shenango St., Greenville, Pa.

Salyer, John M.----------Fitzsimons Hospital, Denver, Colo.
Sanes, Gilmore M.----------3500 Fifth Ave., Pittsburgh 13, Pa.
Schafer, Paul W.----------c/o Message Center, Walter Reed Army Hospital, Washington, D. C.
Seiler, Hawley H.----------517 Bayshore Blvd., Tampa 6, Fla.
Seybold, William D.----------Hermann Professional Bldg., Houston, Texas
Shipman, Sidney----------490 Post St., San Francisco, Calif.
Simpson, H. Murray----------292 Queen's Ave., London, Ont.
Skinner, A. M.----------Homer Folks Tuberculosis Hospital, Oneonta, N. Y.
Snyder, Howard E.----------103½ E. Ninth Ave., Winfield, Kan.
Snyder, John M.----------1236 Moffitt Ave., Bethlehem, Pa.
Starkey, George W. B.----------319 Longwood Ave., Boston 15, Mass.
Stayman, Joseph----------8815 Germantown Ave., Philadelphia 18, Pa.
Stenstrom, John D.----------2390 Bowker Ave., Victoria, B. C.
Sullivan, Herbert J.----------Medical Arts Bldg., Hamilton, Ont.
Swenson, Orvar----------300 Longwood Ave., Boston, Mass.

Taber, Rodman E.----------Henry Ford Hospital, Detroit 2, Mich.
Taylor, Frederick H.----------1012 Kings Drive, Charlotte, N. C.
Templeton, John Y., III----------1025 Walnut St., Philadelphia 7, Pa.
Terrill, Frank I.----------Montana State Tuberculosis Sanatorium, Deer Lodge, Mont.
Test, Frederick C., II----------20252 Meyers Road, Detroit 35, Mich.
Thomas, Gordon W.----------Int. Grenfell Association, St. Anthony, Newfoundland
Tillou, Donald J.----------311 W. Church St., Elmira, N. Y.
Tricerri, Fernando E.----------3, Chemin de Mornex, Lausanne, Switzerland

Valle, A. R.----------U.S.P.H.S. Hospital, Detroit, Mich.
Van Fleit, William E.----------Emory University Hospital, Emory University, Ga.
Veal, J. Ross----------3560 Appleton St., N.W., Washington, D. C.

Walker, George R.----------5 Beach St., Sudbury, Ont.
Walkup, Harry E.----------Veterans Adm. Hospital, Oteen, N. C.
Watkins, David H.----------Denver General Hospital, Denver 4, Colo.
Watkins, Elton, Jr.----------300 Longwood Ave., Boston 15, Mass.
Whiteside, William C.----------415 Medical Arts Bldg., Victoria, B. C.
Wilson, John L.----------Dept. of Surgery, American University of Beirut, Beirut, Lebanon
Witmer, Robert H.----------126 East Chestnut St., Lancaster, Pa.
Wolff, William I.----------30 Central Park South, New York 19, N. Y.

## Senior Members

Allen, Duff S.----------18 S. Kingshighway, St. Louis 8, Mo.
Amberson, J. B.----------Bellevue Hospital, New York, N. Y.

# ANNUAL MEETING NOTES

The Thirty-seventh Annual Meeting of The American Association for Thoracic Surgery was held in Chicago, Illinois, on May 4, 6, and 7, 1957. There were 1,001 physicians who registered for the scientific sessions. In addition, there were 182 wives present.

As a mark of esteem and appreciation for the close contact that has been maintained between The American Association for Thoracic Surgery and the Society of Thoracic Surgeons of Great Britain and Ireland, the latter group proffered a Presidential Badge and Chain of Office. In an appropriate ceremony, held immediately at the conclusion of the Business Session on Saturday, May 4, 1957, Mr. George A. Mason, Vice President of the Society of Thoracic Surgeons of Great Britain and Ireland, presented this gift to our Association. Mr. J. Leigh Collis, the Honorary Secretary, placed the ornament about the neck of Dr. Cameron Haight, the President. The other distinguished representatives were Professor R. S. Pilcher, Mr. Vernon C. Thompson, and Mr. Oswald S. Tubbs.

At the conclusion of the Scientific Session on Tuesday afternoon, May 7, the precedent was established wherein this Presidential Badge and Chain of Office was vested upon the incoming President by the retiring officer of similar rank.

The problems presented by the rapid growth of this Association were studied at great length by an especially appointed committee of which Dr. Edward M. Kent was the Chairman. The proposals advanced in the report of this committee were thoroughly discussed by the membership in Executive Session. In view of the crucial character of any such decision this committee was requested to pursue further its investigation and report the results of such deliberations at a future date.

The following changes in membership status were voted by the Association during its annual Executive Session:

The following Active members were elevated to Senior Status:

Robert G. Bloch
Louis R. Davidson
Harry R. Decker
Louis F. Knoepp
Leon J. Leahy
Herbert Willis Meyer
Richmond L. Moore
M. Dawson Tyson
Joseph A. Weinberg

The following Associate members were elected to Active membership:

Ralph D. Alley
Frank S. Ashburn
Frank Bortone
Garrett M. Brownrigg
John S. Chambers, Jr.
John G. Chesney
George H. A. Clowes, Jr.
David A. Cooper
Oscar Creech, Jr.
Albert C. Daniels
DeWitt C. Daughtry
Norman C. Delarue
Emerson H. Drake
F. Henry Ellis, Jr.
William B. Ford
Theodore R. Hudson
Gerard A. P. Hurley
Elliott S. Hurwitt
Fred J. Jarvis
James H. Johnston, Jr.
John L. Keeley
James R. Laurey
Sanford E. Leeds
William P. Longmire, Jr.
Waldo O. Mills
Emmanuel M. Papper
Richard M. Peters
Beatty H. Ramsey
Charles T. Read
Irving A. Sarot
Joseph E. Strode
William R. Waddell
James H. Walker
Watts R. Webb
Julius L. Wilson
Thomas B. Wiper

Murphy, James D.------------U.S. Veterans Adm. Hospital, Baltimore 18, Md.
Myers, J. Arthur------------730 La Salle Bldg., Minneapolis, Minn.

Neuhof, Harold------------Box 198, Huntington Road, Stratford, Conn.
Nixon, James W.------------1121 Nix Professional Bldg., San Antonio, Texas

O'Brien, Edward J.------------307 David Whitney Bldg., Detroit 26, Mich.
Ornstein, George------------965 Fifth Ave., New York, N. Y.

Packard, Edward N.------------142 Park Ave., Saranac Lake, N. Y.
Pickhardt, Otto C.------------66 East 79th St., New York, N. Y.

Rienhoff, William F., Jr.------------1201 N. Calvert St., Baltimore, Md.
Rigler, Leo G.------------University Hospitals, Minneapolis 14, Minn.
Ross, Dudley E.------------St. Adolphe de Howard, P.Q., Canada

Smith, David T.------------Duke University, Durham, N. C.

Thorburn, Grant------------1602 West Genessee St., Flint, Mich.
Tucker, Gabriel------------250 South 19th St., Philadelphia, Pa.
Tyson, M. Dawson------------Hitchcock Clinic, Hanover, N. H.

Van Allen, Chester M.------------State Hospital, Bikaner, Rajputana, India

Wangensteen, Owen H------------University Hospitals, Minneapolis, Minn.
Weinberg, Joseph A.------------Veterans Adm. Hospital, Long Beach, Calif.
Welles, Edward S.------------20 Church Street, Saranac Lake, N. Y.

## Members Deceased

Carl Eggers
Evarts A. Graham
Felix P. Miller
Harlan F. Newton
Wyman Whittemore

# INDEX TO VOLUME 33

A

Actinomyces bovis, chronic constrictive pericarditis due to (Hart and Pierce), 730

Adkins, Paul C. (with Bauersfeld, S. Richard, and Kent, Edward M.), Patent ductus arteriosus in infancy, 123

Allison, P. R. (with Wooler, G. H., and Gunning, A. J.), Esophagojejunogastrostomy, 738

American Association for Thoracic Surgery, Constitution and By-Laws, 138
  Program, 37th Annual Meeting, 563

Anesthetic agents, two new topical, Dyclone and Cyclaine, evaluation of (Hughes, Mobley, Bills, Burwell, and Pate), 685

Aneurysm, dissecting, surgical treatment of (Gilman and Bailey), 670

Aneurysms, chronic (healed) dissecting (Prior, Burian, and Perl), 213

Angiocardiographic demonstration of superior vena caval constriction in constrictive pericarditis (Lyons, Minnis, and Griffin), 305

Angiocardiography, contributions of, surgical management of large intrathoracic goiters (Miscall, Noland, Finby, and Steinberg), 637
  visualization by, arteriovenous fistula of right internal mammary vessels following radical mastectomy (Glenn and Steinberg), 719

Aortic isthmus, congenital absence of, report of case with successful surgical repair (Merrill, Webster, and Samson), 311

"Arterialized" venous blood for determination of changes in arterial $O_2$ and $CO_2$ tensions following hyperventilation, use of (Brook, Knight, and Cooper), 412

Arteriovenous fistula of right internal mammary vessels following radical mastectomy, visualization by angiocardiography (Glenn and Steinberg), 719

Artery perfusion during total by-pass of heart, coronary and carotid (Kay, Gaertner, Isaacs, and Dever), 526

Artificial hibernation, hemodynamic and electrocardiographic studies at normal temperature and during, experimental interatrial septal defect (de Carvalho Pinto), 585

Austin, R. Reed (with Blair, Emil, Blount, S. Gilbert, Jr., and Swan, Henry), Study of cardiovascular changes during cooling and rewarming in human subjects undergoing total circulatory occlusion, 707

Autotransplantation of spleen into thorax (Skinner and Hurteau), 807

B

Bailey, Charles P., Discussion of mitral insufficiency, 120
  (with Gilman, Richard A.), Surgical treatment of dissecting aneurysm, 670
  (with Morse, Dryden P.), Mitral commissurotomy performed from the right side, [illegible]7

Balogh, Gyula (with Dimitrov Szokodi, Daniel, and Husveti, Alexander), Lung denervation in the therapy of intractable bronchial asthma, 166

Bauersfeld, S. Richard (with Adkins, Paul C., and Kent, Edward M.), Patent ductus arteriosus in infancy, 123, 134

Baum, George (with Schaff, Burnett), Tracheal bronchus, 282

Bilateral resection with space-diminishing procedure for pulmonary tuberculosis, simultaneous (Bjork), 617

Bills, R. J. (with Hughes, F. A., Mobley, J. D., Burwell, J. R., and Pate, J. W.), Evaluation of two new topical anesthetic agents, Dyclone and Cyclaine, 685

Birath, G. (with Swenson, E. W.), Resistance to air flow in bronchospirometric catheters, 275

Bishop, Clarence A., Cancer of lung, analysis and evaluation of 100 consecutive cases, 330

Bjork, Viking Olov, Lobectomy for pulmonary tuberculosis, analysis of 301 cases, 754
  simultaneous bilateral resection with space-diminishing procedure for pulmonary tuberculosis, 617

Black, Harrison, Discussion of mitral insufficiency, 117

Blair, Emil (with Austin, R. Reed, Blount, S. Gilbert, Jr., and Swan, Henry), Study of cardiovascular changes during cooling and rewarming in human subjects undergoing total circulatory occlusion, 707

The following candidates were elected to Associate membership:

Thomas G. Baffes, Chicago, Ill.
James S. Battersby, Indianapolis, Ind.
Martin Bergmann, St. Louis, Mo.
Philip E. Bernatz, Rochester, Minn.
William G. Cahan, New York, N. Y.
Richard G. Connar, Tampa, Fla.
John F. Dammann, Charlottesville, Va.
Robert A. DeBord, Peoria, Ill.
Josiah Fuller, Duluth, Minn.
William W. L. Glenn, New Haven, Conn.
Laurence K. Groves, Cleveland, Ohio
Owen Gwathmey, Washington, D. C.
Joseph M. Hanner, San Diego, Calif.
Edward J. Jahnke, Jr., Washington, D. C.
Robert W. Jamplis, Palo Alto, Calif.
Robert J. Jensik, Chicago, Ill.
Frank R. Johnston, Winston-Salem, N. C.
Robert P. McBurney, Memphis, Tenn.
Peter V. Moulder, Chicago, Ill.
Wilford B. Neptune, Boston, Mass.
John S. Paul, Martinsburg, W. Va.
John W. Polk, Springfield, Mo.
Herbert T. Ransdell, Jr., Louisville, Ky.
David H. Watkins, Denver, Colo.

Cohen, Morley (with Warden Herbert L DeWall, Richard A, Varco, Richard L, and Lillehei C Walton), Surgical pathologic classification for isolated ventricular septal defects and for those in Fallot's tetralogy based on observations made on 120 patients during repair under direct vision, 21
Congenital absence of aortic isthmus, report of case with successful surgical repair (Merrill, Webster, and Samson), 311
Constitution and By Laws, American Association for Thoracic Surgery, 138
Cooley, Denton A Discussion of classification for ventricular septal defects, 44
Cooley, Jack C (with Moser, Fritz H and Hedberg, Gustaf A), Results of pulmonary resection in treatment of tuberculosis, evaluation of 201 consecutive resections 383
Cooper, Philip (with Brook, David L, and Knight, James B, Jr), Use of "arterialized" venous blood for determination of changes in arterial $O_2$ and $CO_2$ tensions following hyperventilation, 412
Coronary occlusion, acute, failure of cardiopericardiopexy to protect pigs against (Gross, Bloomberg, Rosenblatt, Kantrowitz, Schein, and Ferreira), 679
Cyclaine and Dyclone, evaluation of two new topical anesthetic agents (Hughes, Mobley, Bills, Burwell, and Pate), 685

D

Dailey, James E, Discussion of clinical evaluation of decortication, 19
Dammann, Francis, Jr, Discussion of patent ductus arteriosus in infancy, 132
Davila, Julio C (with Glover, Robert P), Treatment of mitral insufficiency by purse string technique, initial clinical application, 75, 119
Davis, Carl, Jr (with Brown, Gordon), Intrathoracic neurofibroma of vagus nerve associated with diaphragmatic hernia, 532
De Carvalho Pinto, Virgilio A, Experimental interatrial septal defect hemodynamic and electrocardiographic studies at normal temperature and during artificial hibernation, 585
Decortication, clinical evaluation of (Waterman, Domm, and Rogers), 1
Dennis, C (with Kernan, M C, Newman, M M, Levowitz, B S, and Stuckey, J H), Prosthesis to replace mitral valve, 698
Derbyshire, A J (with Dodrill, F D, Marshall, Norman, Nyboer, Jan, Hughes C H, and Stearns, A B), Use of heart lung apparatus in human cardiac surgery, 60
Dever Richard (with Kay, Jerome Harold, Gaertner, Robert A, and Isaacs, James P), Coronary and carotid artery perfusion during total bypass of heart, 526
DeWall Richard A (with Warden, Herbert E, Cohen Morley, Varco, Richard L, and Lillehei, C Walton), Surgical pathologic classification for isolated ventricular septal defects and for those in Fallot's tetralogy based on observations made on 120 patients during repair under direct vision, 21
Diaphragm for obliteration of dead space following pulmonary resection, transplantation of (Burdette), 803
Diaphragmatic hernia, intrathoracic neurofibroma of vagus nerve associated with (Davis and Brown), 532
Dickinson, Daniel W (with Kipp, Harold A) Solitary benign neurofibroma of lung coexistent with pulmonary tuberculosis, 551
Dimitrov Szokodi, Daniel (with Husveti, Alexander, and Balogh, Gyula), Lung denervation in the therapy of intractable bronchial asthma, 166
Diverticula, pulsion pharyngeal (Ingram and Foster), 287
Dodrill, F D (with Marshall, Norman, Nyboer, Jan, Hughes, C H, Derbyshire, A J and Stearns, A B), Use of heart lung apparatus in human cardiac surgery, 60, 74
Domm, Sheldon E (with Waterman, David H and Rogers, William K), Clinical evaluation of decortication, 1
Donald, David E (with Kirklin, John W, Harshbarger, Harry G, and Edwards, Jesse E), Surgical correction of ventricular septal defect anatomic and technical considerations, 45
Dyclone and Cyclaine, evaluation of two new topical anesthetic agents (Hughes, Mobley, Bills, Burwell, and Pate), 685

E

Edgcomb, John H (with Kaiser, George C, and Kay, Jerome Harold), Ventricular fibrillation, 537
Edwards, Jesse E (with Kirklin, John W, Harshbarger, Harry G, and Donald, David E), Surgical correction of ventricular septal defect, anatomic and technical considerations, 45
Eggers, Carl, In memoriam, 423
Electrical pacemaker stimuli on human heart, effects of external (Shafiroff and Linder), 544
Emerson, Ernest B, Jr, Ribbon esophagoscopes, 821
Esophagojejunogastrostomy (A [illegible] er, and Gunning), 738
Esophagoscope, rib [illegible]

Blalock, Alfred, Discussion of surgery for ventricular septal defect, 59
Bloomberg, Allan E. (with Gross, Harry, Rosenblatt, Morton, Kantrowitz, Adrian, Schein, Clarence J., and Ferreira, Ruthven), Failure of cardio-pericardiopexy to protect pigs against acute coronary occlusion, 679
Blount, S. Gilbert, Jr. (with Blair, Emil, Austin, R. Reed, and Swan, Henry), Study of cardiovascular changes during cooling and rewarming in human subjects undergoing total circulatory occlusion, 707
Bosher, Lewis H., Discussion of mitral insufficiency, 118
Bowers, Warner F. (with Hewlett, Thomas H., and Puglisi, Anthony J.), Bronchospasm in bronchography, 609
Bowes, Donald E. (with Kirklin, John W., and Swan, H. J. C.), Hemodynamic effects of large atrial septal defects, 350
Bronchi, effect of preoperative chemotherapy on incidence of tuberculous lesions in (Gordon), 399
Bronchial arteries of dog, anatomy of (Notkovich), 242
  asthma, intractable, lung denervation in the therapy of, 166
  epithelium, with special reference to carcinoma of lung, histopathologic observations on (Ryan, McDonald, and Clagett), 264
Bronchogenic carcinoma, involvement of thoracic wall in (Gronqvist, Clagett, and McDonald), 487
Bronchography, bronchospasm in (Hewlett, Puglisi, and Bowers), 609
Bronchopulmonary sequestration; clinical aspects with report of six cases (Pinney and Salyer), 791
Bronchoscopic table designed for office use (Mangiardi), 557
Bronchospasm in bronchography (Hewlett, Puglisi, and Bowers), 609
Bronchospirometric catheters, resistance to air flow in (Swenson and Birath), 275
Bronchus, lipoma of, report of case (Ochsner, LeJeune, and Ochsner), 371
  tracheal (Schaff and Baum), 282
Brook, David L. (with Knight, James B., Jr., and Cooper, Philip), Use of "arterialized" venous blood for determination of changes in arterial $O_2$ and $CO_2$ tensions following hyperventilation, 412
Brown, Gordon (with Davis, Carl, Jr.), Intrathoracic neurofibroma of vagus nerve associated with diaphragmatic hernia, 532
Buechner, Howard A. (with Ziskind, Morton M., and Strug, Lawrence H.), Pneumoperitoneum as space-occupying procedure in conjunction with pulmonary resection, 229
Buran, Robert T. (with Prior, John T., and Perl, Theodore), Chronic (healed) dissecting aneurysms, 213
Burdette, Walter J., Transplantation of diaphragm for obliteration of dead space following pulmonary resection, 803
Burwell, J. R. (with Hughes, F. A., Mobley, J. D., Bills, R. J., and Pate, J. W.), Evaluation of two new topical anesthetic agents: Dyclone and Cyclaine, 685
Butler, Ethan Flagg, In memoriam, Carl Eggers, 423

C

Cancer of lung, analysis and evaluation of 100 consecutive cases (Bishop), 330
Carcinoma of lung, histopathologic observations on bronchial epithelium with special reference to (Ryan, McDonald, and Clagett), 264
  of superior mediastinal and cervical segments of esophagus, anterior approach to (Waddell and Scannell), 663
Cardia, incompetence of (Stensrud), 749
Cardiac surgery, use of heart-lung apparatus in human (Dodrill, Marshall, Nyboer, Hughes, Derbyshire, and Stearns), 60
Cardio-pericardiopexy to protect pigs against acute coronary occlusion, failure of (Gross, Bloomberg, Rosenblatt, Kantrowitz, Schein, and Ferreira), 679
Cardiovascular changes during cooling and rewarming in human subjects undergoing total circulatory occlusion, study of (Blair, Austin, Blount, and Swan), 707
Chambers, John S., Discussion of clinical evaluation of decortication, 17
Chemotherapy on incidence of tuberculous lesions in bronchi, effect of preoperative (Gordon), 399
Cincotti, John J. (with Siebens, Arthur A., Grant, Austin R., Kent, Donald C., and Klopstock, Robert), Pulmonary cystic disease: physiologic studies and results of resection, 185
Circulatory occlusion, total, study of cardiovascular changes during cooling and rewarming in human subjects undergoing (Blair, Austin, Blount, and Swan), 707
Clagett, O. Theron (with Gronqvist, Y. K. J., and McDonald, John R.), Involvement of thoracic wall in bronchogenic carcinoma, 487
  (with Jackson, Robert C., and McDonald, John R.), Pericardial fat necrosis, report of three cases, 723
  (with Ryan, Robert F., and McDonald, John R.), Histopathologic observations on bronchial epithelium with special reference to carcinoma of lung, 264

Holmes, C Leon, Pectus excavatum, surgical technique new form of external traction to elevated sternum 321
Hughes, C H (with Dodrill, F D Marshall, Norman Nyboer, Jan, Derbyshire A J, and Stearns, A B ), Use of heart lung apparatus in human cardiac surgery, 60
Hughes, F A (with Bills, R J, Burwell J R and Pate, J W ), Evaluation of two new topical anesthetic agents Dyclone and Cyclaine, 685
Hurt, Raymond, Discussion of patent ductus arteriosus in infancy, 133
Hurteau, Wm W (with Skinner, Edward F ), Autotransplantation of spleen into thorax 807
Husveti, Alexander (with Dimitrov Szokodi, Daniel, and Balogh, Gyula) Lung denervation in the therapy of intractable bronchial asthma, 166
Hypoplasia according to side affected, note on prognosis of pulmonary agenesis (Schaffer and Rider), 379
Hyperventilation use of "arterialized" venous blood for determination of changes in arterial O and CO, tensions following (Brook, Knight and Cooper), 412

I

Ingram, Ivan N (with Foster R P ), Pulsion pharyngeal diverticula, 287
Interatrial septal defect, experimental hemodynamic and electrocardiographic studies at normal temperature and during artificial hibernation (de Carvalho Pinto), 585
Intracranial metastasis in lung cancer detection of, comparative study of neurological examination and electroencephalogram (Smith), 814
Intrathoracic goiters, large, surgical management of, contributions of angio[illegible] Noland,
lipoma [illegible], 785
neurofibroma of vagus nerve associated with diaphragmatic hernia (Davis and Brown), 5[illegible]2
Isaacs, James P (with Kay, Jerome Harold, Gaertner, Robert A, and Dever, Richard), Coronary and carotid artery perfusion during total by pass of heart, 526

J

Jackson, Robert C (with Clagett, O Theron and McDonald, John R ), Pericardial fat necrosis, report of three cases, 723

K

Kaiser, George C (with Edgcomb, John H, and Kay Jerome Harold), Ventricular fibrillation, 537
Kantrowitz, Adrian (with Gross, Harry, Bloomberg, Allan E, Rosenblatt Morton, Schein, Clarence J, and Ferreira, Ruthven), Failure of cardiopericardiopexy to protect pigs against acute coronary occlusion, 679
Kay, Earle B, Discussion of mitral insufficiency 119
Kay, Jerome Harold (with Gaertner Robert A, Isaacs James P, and Dever, Richard), Coronary and carotid artery perfusion during total by pass of heart, 526
(with Kaiser, George C, and Edgcomb, John H ), Ventricular fibrillation, 7
Kemler, [illegible] Leonard Discussion of clinical evaluation of decortication 18
Kennedy John H (with Spencer Frank C ) [illegible] wounds of heart report of two patients with unusual features 361
Kent, Donald C (with Siebens, Arthur A Grant Austin R Klopstock, Robert and Brown, John J) Pulmonary cystic disease physiologic studies and results of resection, 185
Kent Edward M (with Bauersfeld, S Richard and Adkins Paul C ), Patent ductus arteriosus in infancy, 123
Kernan M C (with Newman M M, Levowitz B S, Stuckey, J H and Dennis [illegible]), Prosthesis to replace mitral valve 698
Kipp, Harold A (with Dickinson Daniel W ) Solitary benign neurofibroma of lung coexistent with pulmonary tuberculosis 551
Kirklin John W (with Bowes Donald E, and Swan, H J C ), Hemodynamic effects of large atrial septal defects, 830
(with Harshbarger Harry G, Donald, David F, and Edwards Jesse E ), Surgical correction of ventricular septal defect anatomic and technical considerations 45 59
Klopstock Robert (with Siebens, Arthur A Grant Austin R, Kent Donald C, and Cincotti John J ) Pulmonary cystic disease physiologic studies and results of resection, 185
Knight, James B, Jr (with Brook David I and Cooper, Philip), Use of "arterialized" venous blood for determination of changes in arterial O and CO tensions following hyperventilation, 412
Kovach, John C (with Morales, George), Clinical significance of oxygen removal, 690

L

Leiomyoma of trachea case report (Green and Winn), 237
LeJeune, Francis E (with Ochsner, Seymour and Ochsner, Alton), Lipoma of bronchus, report of case, 371

Esophagus, anterior approach to carcinoma of superior mediastinal and cervical segments of (Waddell and Scannell), 663

F

Fallot's tetralogy, surgical-pathologic classification for isolated ventricular septal defects and for those in, based on observations made on 120 patients during repair under direct vision (Warden, DeWall, Cohen, Varco, and Lillehei), 21
Ferreira, Ruthven (with Gross, Harry, Bloomberg, Allan E., Rosenblatt, Morton, Kantrowitz, Adrian, and Schein, Clarence J.), Failure of cardio-pericardiopexy to protect pigs against acute coronary occlusion, 679
Finby, Nathaniel (with Miscall, Laurence, *Nolan, Richard B., and Steinberg,* Israel), Surgical management of large intrathoracic goiters; contributions of angiocardiography, 637
5-Hydroxytryptamine (Serotonin) and its relationship to carcinoid-cardiovascular syndrome and Rendu-Osler-Weber's syndrome (Sparks and Tombridge), 401
Foster, R. P. (with Ingram, Ivan N.), Pulsion pharyngeal diverticula, 287
Fraser, Kenneth, Subphrenic abscess, 776

G

Gaertner, Robert A. (with Kay, Jerome Harold, Isaacs, James P., and Dever, Richard), Coronary and carotid artery perfusion during total by pass of heart, 526
Gibbon, John H., Jr., Discussion of heart-lung apparatus in human cardiac surgery, 73
Gilman, Richard A. (with Bailey, Charles P.), Surgical treatment of dissecting aneurysm, 670
Glenn, Frank (with Steinberg, Israel), Arteriovenous fistula of right internal mammary vessels following radical mastectomy: visualization by angiocardiography, 719
Glover, Robert P. (with Davila, Julio C.), Treatment of mitral insufficiency by purse-string technique, initial clinical application, 75, 120
Gordon, Wally, Effect of preoperative chemotherapy on incidence of tuberculous lesions in bronchi, 399
Graham, Evarts A., In memoriam, 562
Grant, Austin R. (with Siebens, Arthur A., Kent, Donald C., Klopstock, Robert, and Cincotti, John J.), Pulmonary cystic disease: physiologic studies and results of resection, 185
Greer, Allen E. (with Winn, George L.), Leiomyoma of trachea, case report, 237
Griffin, E. Harrison (with Minnis, J. F., Jr.), Pectus excavatum: survey and suggestion for maintenance of correction, 625
  (with Lyons, Harold A., and Minnis, Jesse), Angiocardiographic demonstration of superior vena caval constriction in constrictive pericarditis, 305
Gronqvist, Y. K. J. (with Clagett, O. Theron, and McDonald, John R.), Involvement of thoracic wall in bronchogenic carcinoma, 487
Gross, Harry (with Bloomberg, Allan E., Rosenblatt, Morton, Kantrowitz, Adrian, Schein, Clarence J., and Ferreira, Ruthven), Failure of cardiopericardiopexy to protect pigs against acute coronary occlusion, 679
Gunning, A. J. (with Allison, P. R., and Wooler, G. H.), Esophagojejunogastrostomy, 738

H

Haas, Lewis L. (with Harvey, Roger A., and Melchor, Charles F.), Radiation management of apical lung tumors, 496
Hara, M. (with Pierce, J. A.), Chronic constrictive pericarditis due to Actinomyces bovis, 730
Harshbarger, Harry G. (with Kirklin, John W., Donald, David E., and Edwards, Jesse E.), Surgical correction of ventricular septal defect, anatomic and technical considerations, 45
Harvey, Roger A. (with Haas, Lewis L., and Melchor, Charles F.), Radiation management of apical lung tumors, 496
Head, Jerome R., Letters to the editor, 137
Heart, coronary and carotid artery perfusion during total by-pass of (Kay, Gaertner, Isaacs, and Dever), 526
  effects of external electrical pacemaker stimuli on (Shafiroff and Linder), 544
  war wounds of, report of two patients with unusual features (Spencer and Kennedy), 361
Heart-lung apparatus in human cardiac surgery, use of (Dodrill, Marshall, Nyboer, Hughes, Derbyshire, and Stearns), 60
Hedberg, Gustaf A. (with Cooley, Jack D., and Moser, Fritz H.), Results of pulmonary resection in treatment of tuberculosis, evaluation of 201 consecutive resections, 383
Hemodynamic effects of large atrial septal defects (Bowes, Kirklin, and Swan), 350
Hewlett, Thomas H. (with Puglisi, Anthony J., and Bowers, Warner F.), Bronchospasm in bronchography, 609
Histopathologic observations on bronchial epithelium with special reference to carcinoma of lung (Ryan, McDonald, and Clagett), 264

Myers, Gordon D (with Slick, John L), Review of 34 consecutive cases of pneumonectomy, with and without concomitant thoracoplasty in treatment of tuberculosis, 567

N

Nanson, Eric M, Thoracic meningocele associated with neurofibromatosis, 650
Necrosis pericardial fat report of three cases (Jackson, Clagett, and McDonald), 723
Neurofibroma of lung coexistent with pulmonary tuberculosis, solitary benign (Dickinson and Kipp), 551
Neurofibromatosis, thoracic meningocele associated with (Nanson), 650
Newman M M (with Kernan, M C, Levowitz, B S, Stuckey, J H, and Dennis, C), Prosthesis to replace mitral valve, 698
  discussion of mitral insufficiency, 117
Nichols, Henry T, Mitral insufficiency treatment by polar cross fusion of mitral annulus fibrosus, 102, 121
Nolan, Richard B (with Miscall, Laurence, Finby, Nathaniel, and Steinberg, Israel), Surgical management of large intrathoracic goiters, contributions of angiocardiography, 637
Notkovich, Hilel, Anatomy of bronchial arteries of dog, 242
Nyboer, Jan (with Dodrill F D, Marshall, Norman, Hughes C H, Derbyshire A J, and Stearns, A B), Use of heart lung apparatus in human cardiac surgery, 60

O

Ochsner, Seymour (with LeJeune, Francis E and Ochsner, Alton), Lipoma of bronchus, report of case, 371
Oxygen removal clinical significance of (Kovach and Morales), 690

P

Parsons, Willard H (with Williams, William F), Intrathoracic lipomas, 785
Pate, J W (with Hughes, F A, Mobley, J D, Bills, R J, and Burwell, J R), Evaluation of two new topical anesthetic agents Dyclone and Cyclaine, 685
Patent ductus arteriosus in infancy (Bauersfeld, Adkins, and Kent), 123
Pectus excavatum, surgical technique new form of external traction to elevated sternum, 321
  survey and suggestion for maintenance of correction (Griffin and Minnis), 625
Pericardial fat necrosis, report of three cases (Jackson, Clagett, and McDonald), 723
Pericarditis, constrictive, angiocardiographic demonstration of superior vena caval constriction in (Lyons, Minnis, and Griffin), 305
  chronic, due to Actinomyces bovis (Hart and Pierce), 730
Perl, Theodore (with Prior, John T, and Buran Robert T), Chronic (healed) dissecting aneurysms, 213
Pierce, J A (with Hart, M), Chronic constrictive pericarditis due to Actinomyces bovis, 730
Pinney, Charles T (with Salyer, John M), Bronchopulmonary sequestration, clinical aspects with report of six cases, 791
Pleural space residual, fate of, following small resections for pulmonary tuberculosis (Weisel, Thimmig, and Watson), 390
Pneumonectomy with and without concomitant thoracoplasty in treatment of tuberculosis, review of 34 consecutive cases (Slick and Myers), 567
Pneumoperitoneum as space occupying procedure in conjunction with pulmonary resection (Beuchner, Ziskind, and Strug), 229
Polar cross fusion of mitral annulus fibrosus, mitral insufficiency treatment by (Nichols), 102
Prior, John T (with Buran, Robert T, and Perl, Theodore) Chronic (healed) dissecting aneurysms, 213
Prosthesis to replace mitral valve (Kernan, Newman, Levowitz, Stuckey, and Dennis), 698
Puglisi Anthony T (with Hewlett, Thomas H, and Bowers, Warner F), Bronchospasm in bronchography, 609
Pulmonary agenesis and hypoplasia according to side affected (Schaffer and Rider), 379
  cystic disease, physiologic studies and results of resection (Siebens, Grant, Kent, Klopstock, and Cincotti), 185
  resection pneumoperitoneum as space occupying procedure in conjunction with (Buechner, Ziskind, and Strug), 229
    transplantation of diaphragm for obliteration of dead space following (Burdette), 803
    in treatment of tuberculosis, evaluation of 201 consecutive resections (Cooley, Moser, and Hedberg), 383
  tuberculosis, fate of residual pleural space following small resections for (Weisel, Thimmig, and Watson), 390
    immediate postoperative complications of thoracic surgery in (Thomas), 341
    lobectomy for, analysis of 301 cases (Bjork), 754
    simultaneous bilateral resection with space diminishing procedure for (Bjork), 617

Letters to the Editor, 135
Levowitz, B. S. (with Kernan, M. C., Newman, M. M., Stuckey, J. H., and Dennis, C.), Prosthesis to replace mitral valve, 698
Lillehei, C. Walton, Discussion of surgery for ventricular septal defect, 57
  (with Warden, Herbert E., DeWall, Richard A., Cohen, Morley, and Varco, Richard L.), Surgical-pathologic classification for isolated ventricular septal defects and for those in Fallot's tetralogy based on observations made on 120 patients during repair under direct vision, 21
Linder, J. (with Shafiroff, B. G. P.), Effects of external electrical pacemaker stimuli on human heart, 544
Lipoma of bronchus, report of case (Ochsner, LeJeune, and Ochsner), 371
Lobectomy for pulmonary tuberculosis, analysis of 301 cases (Björk), 754
Lung cancer, analysis and evaluation of 100 consecutive cases (Bishop), 330
  detection of intracranial metastasis in; comparative study of neurological examination and electroencephalogram (Smith), 814
  denervation in therapy of intractable bronchial asthma (Dimitrov-Szokodi, Husvéti, and Balogh), 166
  primary lymphosarcoma of (Rose), 254
  tumors, apical, radiation management of (Haas, Harvey, and Melchor), 496
Lymphosarcoma of lung, primary (Rose), 254
Lyons, Harold A. (with Minnis, Jesse, and Griffin, Edwin), Angiocardiographic demonstration of superior vena caval constriction in constrictive pericarditis, 305

M

McDonald, John R. (with Gronqvist, Y. K. J., and Clagett, O. Theron), Involvement of thoracic wall in bronchogenic carcinoma, 487
  (with Jackson, Robert C., and Clagett, O. Theron), Pericardial fat necrosis, report of three cases, 723
  (with Ryan, Robert F., and Clagett, O. Theron), Histopathologic observations on bronchial epithelium with special reference to carcinoma of lung, 264
Mangiardi, Joseph L., Bronchoscopic table designed for office use, 557
Marshall, Norman (with Dodrill, F. D., Nyboer, Jan, Hughes, C. H., Derbyshire, A. J., and Stearns, A. B.), Use of heart-lung apparatus in human cardiac surgery, 60
Mastectomy, radical, arteriovenous fistula of right internal mammary vessels following; visualization by angiocardiography (Glenn and Steinberg), 719
Meade, Richard H., Discussion of clinical evaluation of decortication, 19
  of mitral insufficiency, 119
Melchor, Charles F. (with Haas, Lewis L., and Harvey, Roger A.), Radiation management of apical lung tumors, 496
Merrill, Duane L. (with Webster, Charles A., and Samson, Paul C.), Congenital absence of aortic isthmus, report of case with successful surgical repair, 311
Minnis, J. F., Jr. (with Griffin, E. Harrison), Pectus excavatum: survey and suggestion for maintenance of correction, 625
  (with Lyons, Harold A., and Griffin, Edwin), Angiocardiographic demonstration of superior vena caval constriction in constrictive pericarditis, 305
Miscall, Laurence (with Noland, Richard B., Finby, Nathaniel, and Steinberg, Israel), Surgical management of large intrathoracic goiters; contributions of angiocardiography, 637
Mitral and aortic insufficiency in dogs by transventricular injection and retrograde catheterization, roentgen contrast diagnosis of experimental (Wilder, Moscovitz, and Ravitch), 147
  commissurotomy performed from right side (Bailey and Morse), 427
  insufficiency by purse-string technique, treatment of (Glover and Davila), 75
    treatment by polar cross-fusion of mitral annulus fibrosus (Nichols), 102
  valve, prosthesis to replace (Kernan, Newman, Levowitz, Stuckey, and Dennis), 698
Mobley, J. D. (with Hughes, F. A., Bills, R. J., Burwell, J. R., and Pate, J. W.), Evaluation of two new topical anesthetic agents: Dyclone and Cyclaine, 685
Morales, George (with Kovach, John C.), Clinical significance of oxygen removal, 690
Morse, Dryden P. (with Bailey, Charles P.), Mitral commissurotomy performed from the right side, 427
Moscovitz, Howard L. (with Wilder, Robert J., and Ravitch, Mark M.), Roentgen contrast diagnosis of experimental mitral and aortic insufficiency in dogs by transventricular injection and retrograde catheterization, 147
Moser, Fritz H. (with Cooley, Jack C., and Hedberg, Gustaf A.), Results of pulmonary resection in treatment of tuberculosis, evaluation of 201 consecutive resections, 383
Myasthenia gravis secondary to thymic neoplasm, report of case in which symptoms developed six weeks after total thymectomy, 770

Steinberg, Israel (with Glenn, Frank), Arteriovenous fistula of right internal mammary vessels following radical mastectomy, visualization by angiocardiography, 719
  (with Miscall, Laurence, Nolan, Richard B, and Finby, Nathaniel), Surgical management of large intrathoracic goiters, contributions of angiocardiography, 637
Stensrud, Nils Incompetence of cardia, 749
Stephens, H Brodie, Discussion of patent ductus arteriosus in infancy, 131
Stranahan, Allan, Discussion of clinical evaluation of decortication, 16
Strug Lawrence H (with Buechner, Howard A, and Ziskind, Morton M), Pneumoperitoneum as space occupying procedure in conjunction with pulmonary resection, 229
Stuckey, J H (with Kernan, M C, Newman, M M, Levowitz, B S and Dennis, C), Prosthesis to replace mitral valve, 698
Subphrenic abscess (Fraser), 776
Swan, H J C (with Bowes, Donald E, and Kirklin John W), Hemodynamic effects of large atrial septal defects, 350
Swan, Henry (with Blair, Emil, Austin R Reed, and Blount, S Gilbert Jr), Study of cardiovascular changes during cooling and rewarming in human subjects undergoing total circulatory occlusion 707
Swenson, E W (with Birath, G), Resistance to air flow in bronchospirometric catheters 275
Syndrome, Rendu Osler Weber's, 5 Hydroxytryptamine (Serotonin) and its relationship to carcinoid cardiovascular syndrome and (Sparks and Tombridge), 401

T

Thimmig, Robert F (with Weisel, Wilson and Watson Raymond R), Fate of residual pleural space following small resections for pulmonary tuberculosis, 390
Thomas, David E, Immediate postoperative complications of thoracic surgery in pulmonary tuberculosis, 341
Thoracic meningocele associated with neurofibromatosis (Nanson), 650
  wall in bronchogenic carcinoma, involvement of (Gronqvist, Clagett, and McDonald), 487
Thoracoplasty, concomitant, in treatment of tuberculosis, review of 34 consecutive cases of pneumonectomy with and without (Shek and Myers), 567
Thorax, autotransplantation of spleen into (Skinner and Hurteau), 807
Thymic neoplasm myasthenia gravis secondary to, report of case in which symptoms developed six weeks after total thymectomy, 770
Tombridge, Thomas L (with Sparks, Charles H), 5 Hydroxytryptamine (Serotonin) and its relationship to carcinoid cardiovascular syndrome and Rendu Osler Weber's syndrome, 401
Trachea leiomyoma of, case report (Greer and Winn), 237
Transventricular injection and retrograde catheterization roentgen contrast diagnosis of experimental mitral and aortic insufficiency in dogs by Wilder, Moscovitz, and Ravitch), 47
Tuberculosis results of pulmonary resection in treatment of evaluation of 201 consecutive resections (Cooley, Moser, and Hedberg), 383
  review of 34 consecutive cases of pneumonectomy, with and without concomitant thoracoplasty, in treatment of (Shek and Myers), 567
Tuberculous lesions in bronchi, effect of preoperative chemotherapy on incidence of (Gordon), 399

V

Vagus nerve associated with diaphragmatic hernia intrathoracic neurofibroma of (Davis and Brown), 532
Valle, A R, Discussion of clinical evaluation of decortication 19
Varco, Richard L (with Warden, Herbert E, DeWall, Richard A, Cohen, Morley, and Lillehei C Walton), Surgical pathologic classification for isolated ventricular septal defects and for those in Fallot's tetralogy based on observations made on 120 patients during repair under direct vision, 21
Vena caval constriction, superior, in constrictive pericarditis, angiocardiographic demonstration of (Lyons, Minnis, and Griffin), 305
Ventricular fibrillation (Kaiser, Edgcomb, and Kay), 537
  septal defect, surgical correction of, anatomic and technical considerations (Kirklin, Harshbarger, Donald, and Edwards), 45
    defects isolated, and those in Fallot's tetralogy, surgical pathologic classification for, based on observations made on 120 patients during repair under direct vision (Warden, DeWall, Cohen, Varco, and Lillehei), 21

W

Waddell William R (with Scannell, J Gordon), Anterior approach to carcinoma of superior mediastinal and cervical segments of es

Pulmonary tuberculosis—Cont'd
  solitary benign neurofibroma of lung coexistent with (Dickinson and Kipp), 551
Pulsion pharyngeal diverticula (Ingram and Foster), 287
Purse-string technique, treatment of mitral insufficiency by (Glover and Davila), 75

R

Radiation management of apical lung tumors (Haas, Harvey, and Melchor), 496
Ravitch, Mark M. (with Wilder, Robert J., and Moscovitz, Howard L.), Roentgen contrast diagnosis of experimental mitral and aortic insufficiency in dogs by transventricular injection and retrograde catheterization, 147
Resection, physiologic studies and results of, in pulmonary cystic disease (Siebens, Grant, Kent, Klopstock, and Cincotti), 185
Resections, small, for pulmonary tuberculosis, fate of residual pleural space following (Weisel, Thimmig, and Watson), 390
Rider, Rowland V. (with Schaffer, Alexander J.), Note on prognosis of pulmonary agenesis and hypoplasia according to side affected, 379
Roe, Benson B., Myasthenia gravis secondary to thymic neoplasm, report of case in which symptoms developed six weeks after total thymectomy, 770
Roentgen contrast diagnosis of experimental mitral and aortic insufficiency in dogs by transventricular injection and retrograde catheterization (Wilder, Moscovitz, and Ravitch), 147
Rogers, William K., Discussion of clinical evaluation of decortication, 18
  (with Waterman, David H., and Domm, Sheldon E.), Clinical evaluation of decortication, 1
Rose, A. Hewitt, Primary lymphosarcoma of lung, 254
Rosenblatt, Morton (with Gross, Harry, Bloomberg, Allan E., Kantrowitz, Adrian, Schein, Clarence J., and Ferreira, Ruthven), Failure of cardio-pericardiopexy to protect pigs against acute coronary occlusion, 679
Ryan, Robert F. (with McDonald, John R., and Clagett, O. Theron), Histopathologic observations on bronchial epithelium with special reference to carcinoma of lung, 264

S

Salyer, John M. (with Pinney, Charles T.), Bronchopulmonary sequestration, clinical aspects with report of six cases, 791
Samson, Paul C., Discussion of clinical evaluation of decortication, 18
  (with Merrill, Duane L., and Webster, Charles A.), Congenital absence of aortic isthmus, report of case with successful surgical repair, 311
Scannell, J. Gordon (with Waddell, William R.), Anterior approach to carcinoma of superior mediastinal and cervical segments of esophagus, 663
Schaff, Burnett (with Baum, George), Tracheal bronchus, 282
Schaffer, Alexander J. (with Rider, Rowland V.), Note on prognosis of pulmonary agenesis and hypoplasia according to side affected, 379
Schein, Clarence J. (with Gross, Harry, Bloomberg, Allan E., Rosenblatt, Morton, Kantrowitz, Adrian, and Ferreira, Ruthven), Failure of cardio-pericardiopexy to protect pigs against acute coronary occlusion, 679
Schotz, Seymour, Letters to the editor, 136
Seley, Gabriel, Discussion of clinical evaluation of decortication, 17
Septal defects, large atrial, hemodynamic effects of (Bowes, Kirklin, and Swan), 350
Shafiroff, B. G. P. (with Linder, J.), Effects of external electrical pacemaker stimuli on human heart, 544
Shek, John L. (with Myers, Gordon D.), Review of 34 consecutive cases of pneumonectomy, with and without concomitant thoracoplasty, in treatment of tuberculosis, 567
Siebens, Arthur A. (with Grant, Austin R., Kent, Donald C., Klopstock, Robert, and Cincotti, John J.), Pulmonary cystic disease: physiologic studies and results of resection, 185
Skinner, Edward F. (with Hurteau, Wm. W.), Autotransplantation of spleen into thorax, 807
Smith, James K., Detection of intracranial metastasis in lung cancer; comparative study of neurological examination and electroencephalogram, 814
Sones, F. Mason, Jr., Discussion of patent ductus arteriosus in infancy, 133
Sparks, Charles H. (with Tombridge, Thomas L.), 5-Hydroxytryptamine (Serotonin) and its relationship to carcinoid-cardiovascular syndrome and Rendu-Osler-Weber's syndrome, 401
Spencer, Frank C. (with Kennedy, John H.), War wounds of heart, report of two patients with unusual features, 361
Stearns, A. B. (with Dodrill, F. D., Marshall, Norman, Nyboer, Jan, Hughes, C. H., and Derbyshire, A. J.), Use of heart-lung apparatus in human cardiac surgery, 60

War wounds of heart, report of two patients with unusual features (Spencer and Kennedy), 361

Warden, Herbert E., Discussion of patent ductus arteriosus in infancy, 133

(with DeWall, Richard A., Cohen, Morley, Varco, Richard L., and Lillehei, C. Walton), Surgical-pathologic classification for isolated ventricular septal defects and for those in Fallot's tetralogy based on observations made on 120 patients during repair under direct vision, 21, 44

Waterman, David H. (with Domm, Sheldon E., and Rogers, William K.), Clinical evaluation of decortication, 1, 19

Watson, Raymond R. (with Weisel, Wilson, and Thimmig, Robert F.), Fate of residual pleural space following small resections for pulmonary tuberculosis, 390

Webster, Charles A. (with Merrill, Duane L., and Samson, Paul C.), Congenital absence of aortic isthmus, report of case with successful surgical repair, 311

Weisel, Wilson (with Thimmig, Robert F., and Watson, Raymond R.), Fate of residual pleural space following small resections for pulmonary tuberculosis, 390

Wilder, Robert J. (with Moscovitz, Howard L., and Ravitch, Mark M.), Roentgen contrast diagnosis of experimental mitral and aortic insufficiency in dogs by transventricular injection and retrograde catheterization, 147

Williams, William T. (with Parsons, Willard H.), Intrathoracic lipomas, 785

Winn, George L. (with Greer, Allen E.), Leiomyoma of trachea, case report, 237

Wooler, G. H. (with Allison, P. R., and Gunning, A. J.), Esophagojejunogastrostomy, 738

Z

Ziskind, Morton M. (with Buechner, Howard A., and Strug, Lawrence H.), Pneumoperitoneum [illegible] procedure in [illegible] nary resection, 229